Parasites *of* SWINE

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Worm Parasites *of* Domesticated Animals

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Preface

The object in writing this series of books is to furnish parasitologists and veterinarians with a reasonably complete treatise dealing with the involved subject matter. It attempts to cover all the species of parasitic worms, and incidentally of linguatulids, which have been described from or reported from domesticated animals, and to answer in regard to these worms and in some detail all reasonable questions in regard to nomenclature, hosts, morphology, life history, distribution, pathology, treatment and prophylaxis, so far as information on these topics is available to the writer. No textbook can furnish this information and remain a textbook, and the answers to these questions are only available to specialists with access to extensive libraries. The material in these books has been compiled from numerous sources, the standard text and reference works, such as those of Railliet, Neumann, Neveu-Lemaire, Brumpt, Gedoelst, Fiebiger, and Sluiter, Swellengrebel and Ihle, from numerous monographs, American and foreign, and from the shorter papers published in scientific periodicals or issued by various governments, institutions and societies.

Where facts have been generally known and accepted for many years the authority for statements of such facts is not given, as a rule, in this book. Where they have been recently ascertained or where findings have not been verified by other workers, or where there are contradictory statements, the authority for statements is usually given. While the writer is responsible for many statements in this book, especially those in regard to treatments for parasitic infestations, it is understood, of course, that a book of this character must be essentially a compilation of knowledge, largely based on observations which have not been verified and cannot be verified by the present writer.

Bibliographic citations are not given in this book. There are certain advantages about such citations and certain disadvantages, notable among the disadvantages being the considerable labor of preparing such citations for inclusion and the expense of publishing them. In view of the existence of such excellent catalogues and indices as those of Stiles and Hassall, it has seemed to the writer that little would be lost by omitting such citations in these books.

It is a somewhat difficult task to write for the parasitologist, who commonly wishes technical information, and for the veterinarian, who commonly wishes practical information furnished with as little tech-

nical wording as possible. To accomplish this task without sacrificing technical information in order to make these books more readable for the veterinarian, a certain arrangement of the subject matter has been adopted. It is assumed that as a rule the veterinarian is interested mostly in parasitic species and but little in general in higher groups. The parasites are therefore dealt with in detail as species under their various hosts for the benefit of the veterinarian, and to facilitate finding these species the common names of the parasites and such commonly used scientific names as have fallen into synonomy are given in connection with the correct scientific names and are covered in the index. In this way it should be possible for the veterinarian to find specific parasites regardless of alterations in nomenclature. For the benefit of the parasitologist and such veterinarians as are specially interested in parasitology, dealing with material which is to be identified or concerning which the known technical facts are desired, a taxonomic section, dealing with groups from genera to phyla and including keys down to species, is incorporated in a separate volume to appear as the last of this series.

The field covered by this book is too large for one writer to pass critical judgment on many of the topics, and in general the present writer has accepted new species, genera, etc., as proposed by various writers, regardless of his personal opinion of the new names, except in those fields where a first-hand knowledge or the decided weight of authority warrants passing a critical judgment. New findings may seem highly improbable and yet be true. It is therefore unsafe to reject statements merely because they do not accord with previous concepts, especially where no evidence is supplied for examination and the competency of the authority is unknown. It has been remarked by Leuckart, Cobbold, Rodenwaldt and Roeckemann and others that the field of parasitology is a field of surprises.

Parasites which are of rare and perhaps accidental occurrence or of no known economic importance are accorded as full discussion as available information permits of and the place of these volumes in the field of veterinary parasitology could warrant, since experience shows that we may be mistaken in regarding these parasites as unimportant and because these are the parasites which take excessive amounts of the parasitologist's time when they are presented for identification, owing to their sketchy treatment or omission in most text and reference books and the necessity for a prolonged search through the literature.

The Department of Agriculture in granting permission to write and publish this work stipulates, as in the case of all similar work intended for outside publication on the part of members of its staff, that unpublished work shall not be included in it. This accounts for the fact that this work has no original illustrations. While this arrangement has certain evident disadvantages, it has certain advantages. It obviates the likelihood of publishing figures based on misidentifications of American material presumed to be identical with species described from other countries. As far as possible all species discussed are figured, if figures exist and are available in the published literature. In some cases the only existing figures are old and more or less inadequate; they usually serve some purpose in identification and if they have little other interest, they may have a certain historic interest. They sometimes show that we have no good figures extant for very common parasites.

It is inevitable that there will be certain errors and omissions in a work of this sort. Such works must be published in spite of the certainty of errors or never published. Realizing this the writer will welcome specific criticisms calling attention to errors or omissions. We frequently note such things without troubling the author about them, assuming that he will find them himself. Frequently he does not and they appear in second editions and later ones. It is a service to call the attention of an author to an oversight.

The term swine in this book means *Sus scrofa domestica,* cattle *Bos taurus,* sheep *Ovis aries,* horse *Equus caballus,* dog *Canis familiaris,* and cat *Felis domestica.* Where the common name of other hosts is not sufficiently distinctive the scientific name is usually given.

The writer is indebted to his colleagues and associates in the Zoological Division of the Bureau of Animal Industry for much valuable assistance in connection with the writing and publishing of this book.

MAURICE C. HALL

WORMS IN SWINE

CHAPTER I

Flukes Infesting Swine

AGAMODISTOMUM SUIS Stiles, 1898

The muscle fluke of swine

Synonym.—*Distomum musculorum suis* Duncker, 1896.

Hosts.—Primary: Unknown; secondary: Swine and, apparently, wild boar.

Location.—Free or encysted between the muscle fibres.

Morphology.—*Agamodistomum*: Small, immature flukes 0.5 to 0.7 mm. long by 0.2 mm. wide (Fig. 1). The short intestinal ceca extend past the acetabulum and beyond the middle of the body, but terminate anterior of the primordia of the genital glands.

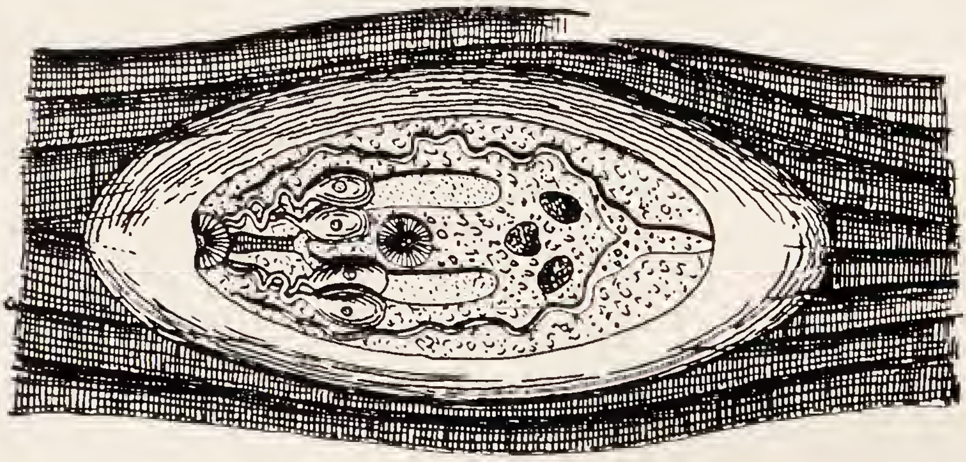


Fig. 1. *Agamodistomum suis.* Fluke encysted in the muscles of swine. Enlarged. From Stiles, 1898, after Leuckart.

Life history.—Unknown. Probably accidentally present in the flesh of swine; their rarity indicates that this is not the true intermediate host of this worm.

Distribution.—Germany and the United States. Stiles is not sure that the form he reported from the United States (at Buffalo, N. Y.) is identical with the form found by Leunis in Saxony and by trichina inspectors elsewhere in Germany, but thinks that it is not improbable. He suggests, however, that they might be erratic *Paragonimus.* Agerth has reported *Agamodistomum*, probably this species, from the wild boar.

Pathology.—These flukes occasion a local reaction with cyst formation, but are so rare and have been found in such small numbers as

to give rise to no known clinical symptoms. They might be mistaken on casual examination for trichinae or sarcosporidia. So far as known, they are of no importance from the standpoint of veterinary practice or meat inspection.

Treatment.—None known.

Prophylaxis.—None known at present owing to our ignorance of the exact identity and life history of the parasite.

FASCIOLA HEPATICA Linnaeus, 1758

The common liver fluke

Synonym.—*Distomum hepaticum* (Linnaeus, 1758) Abildgaard.

Hosts.—Primary: Swine, sheep, cattle and other hosts (See Parasites of Sheep); secondary: Snails (See Parasites of Sheep).

Location.—Biliary canals, bile duct, cystic duct and common duct of primary hosts, and as erratic parasites in the lungs, spleen and heart, also retropleural, retroperitoneal, subcutaneous and elsewhere; in liver of secondary hosts.

Morphology.—Flattened, leaf-like parasites (Fig. 2), about an inch long, as a rule. For details of structure see Parasites of Sheep.

Fig. 2. *Fasciola hepatica.* Entire worm. Ventral view. Natural size. From Stiles, 1898.

Life history.—Involves intermediate stages in snails. For details see Parasites of Sheep.

Distribution.—Widely distributed over the world. Occurs in the United States, especially in the South. Dr. Dalrymple tells me that this fluke is found in perhaps 1 per cent of the swine around Baton Rouge.

Pathology.—The clinical features of hepatic distomatosis in swine have been described by Frenkel. He recognizes 2 types of the disease. One type is gall duct distomatosis and the other parenchymatous distomatosis. The first is characterized by cholangitis, the flukes being present in the gall ducts and gall bladder. The second is an acute, subacute or chronic parenchymatous hepatitis, which may go on to a chronic interstitial hepatitis, and is associated with immature flukes in the liver parenchyma. In the latter condition the capsule of the liver is frequently roughened and adhesions to the peritoneum may occur. Palpation of the liver surface often reveals small inequalities and the

capsule may be distended by small prominences in some places. On section the prominences are found to vary from the size of a hemp seed to that of a hazel nut. In such areas there may be intense hemorrhagic hepatitis or chronic interstitial hepatitis with some degree of encapsulation. Young flukes may be found in some of these areas, the flukes being not far developed beyond the cercaria stage. This form of the disease appears to be commonest among young swine about 6 months old. Considerable quantities of light yellow serous fluid containing threads of fibrin are not uncommonly found in the peritoneal cavity. In the acute stage Frenkel finds ruptures of the capsule and the presence of fibrous exudate in the peritoneal cavity of frequent occurrence.

Treatment.—The use of oleoresin of male fern or kamala is indicated. Presumably the effective dose would be the same as for sheep, since Winslow gives the same dose (one-half drams) for both. This would make the treatment consist of 4 to 6 doses of 1 dram for each 5 kilos of live weight, each dose to be given in the morning of consecutive days, 2 hours before feeding. It would probably be advisable to make the dose for a 100-pound animal the maximum dose for swine.

Prophylaxis.—Provide safe water supplies and keep animals off snail-infested areas, such as wet, swampy pastures. For details see Parasites of Sheep.

FASCIOLOPSIS BUSKI (Lankester, 1857, emend. R. Blanchard, 1888), Odhner, 1902

Busk's intestinal fluke

Synonyms.—*Distoma buskii* Lankester, 1857; *Distomum crassum* Cobbold, 1860, not *Distomum crassum* v. Siebold, 1836; *Distoma buski* Blanchard, 1888; *Fasciolopsis buskii* (Lankester, 1857) Stiles, 1901; *Fasciolopsis fuelleborni* Rodenwaldt, 1909; *Fasciolopsis goddardi* Ward, 1910; *Fasciolopsis spinifera* Brown, 1917. *Fasciolopsis rathouisi* (p. 12) is also regarded as a synonym by some writers.

Hosts.—Primary: Swine, dog, goat and man; secondary: Snails (*Planorbis coenosus* and *Segmentaria largillierti* in Formosa, according to Nakagawa); Stephens states that "the larval stages are said to occur in shrimps," but this is apparently only a hypothesis and at variance with the facts. Atkinson states that cercariae found on prawns had characters strongly suggestive of *Fasciolopsis,* but feeding these to pigs produced no results.

Location.—Intestine of primary host; tissues, especially liver, of secondary host.

Morphology.—*Fasciolopsis*: Body 2.4 to 7 cm. long by 5.5 to 14 mm. wide. Cuticle spiny, the spines easily lost. Ventral sucker larger than oral sucker, the oral about 400μ in diameter and the ventral 1 mm. in small specimens (Fig. 3). Pharynx globular. Intestinal ceca extend to posterior border of body, usually with 2 characteristic curves toward the median line at the anterior margin of the anterior testis and between the 2 testes. Excretory pore on posterior dorsal surface, the median vessel extending to shell gland, dividing to surround it, and uniting again in front of it. Testes much branched and one anterior to the other in the median field. The branching ovary and the compact shell gland are just in front of the anterior testis. The marginal vitellaria extend from the ventral sucker to the posterior margin of the body. Eggs 120 to 130μ long by 77 to 80μ wide.

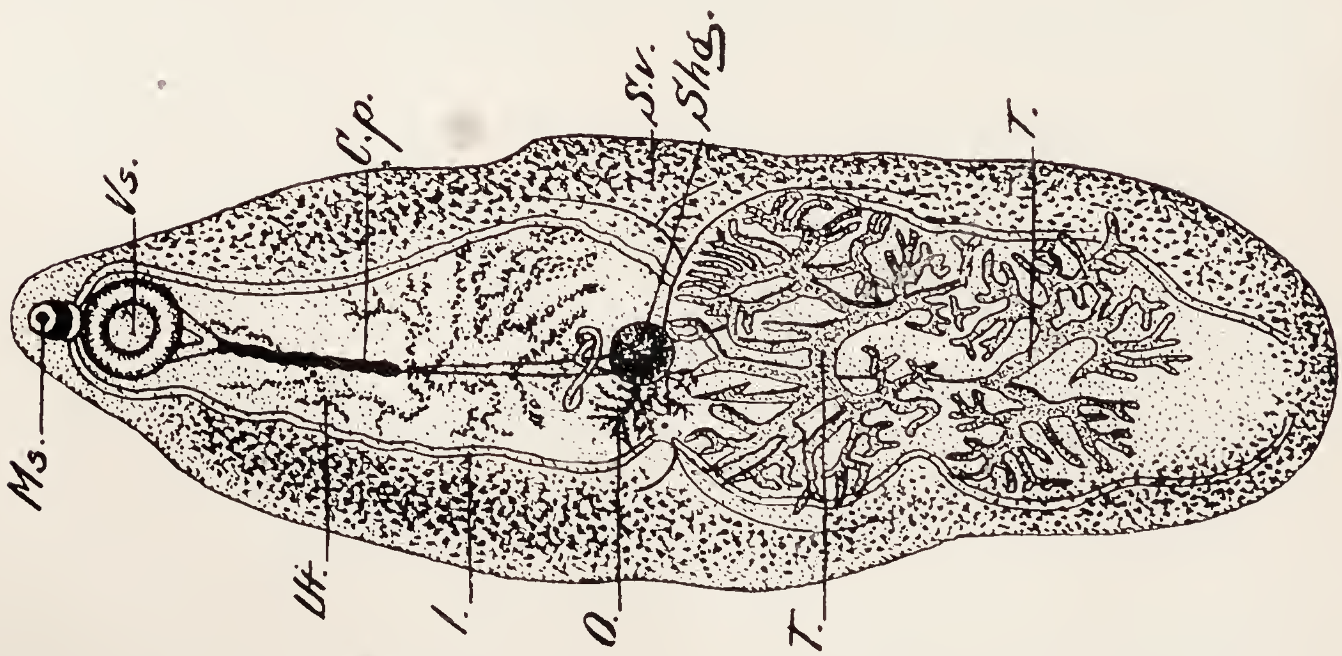


Fig. 3. *Fasciolopsis buski.* Entire worm. Ventral view. Enlarged. From Stephens, 1916, after Odhner. *ms,* oral sucker; *vs,* ventral sucker; *ut,* uterus; *cp,* cirrus pouch; *o,* ovary; *sv,* vitellaria; *shg,* shell gland; *t,* testes.

The cercaria has a distinct bilateral bifurcation of the excretory vesicle.

Life history.—The eggs produced by the flukes pass in the feces and hatch in 2 to 3 weeks at summer temperatures in Formosa, according to Nakagawa. The miracidia enter certain snails and finally the larval development terminates in about 40 days in the production of cercariae which for the most part encyst on water mosses. When these encysted cercariae are swallowed by a suitable host (swine, dog or man), they develop to adults in the intestine, egg production occurring in about a month. Barlow swallowed a number of live flukes

of this species, one fluke lodging as a parasite and remaining a year, when it was expelled by beta-naphthol. Barlow finds certain differences in the developmental stages of *Fasciolopsis* of man from those described for the pig form and thinks they may be different. He says human infestation takes place from eating water chestnut or water redling on which cercariae encyst. No cases in pigs have been found in the areas of greatest human infestation around Shaohsing, China.

Distribution.—India, Siam, China, Formosa, Assam and Sumatra. This fluke is common in swine in Hong Kong, and occurs in 6 per cent of the swine at Hanoi according to Stephens.

Pathology.—According to Odhner, this parasite in man occasions bloody stools and a high fever with a condition of apathy. The flukes are blood suckers and give rise to lesions of the mucosa. Barlow notes that the worms evidently give rise to a high grade toxic condition, sometimes characterized by profound emaciation and sometimes by edema and ascites. Anemia and chronic diarrhea are reported as symptoms, edema being prominent in the late stages. Patients often complain that they can feel the movements of the worms in them. In severe cases, of which there are many in China, worms may be present by thousands. The disease frequently terminates fatally and sometimes wipes out entire families.

While the condition in swine has received little close study, apparently, it is nevertheless probable from the condition produced in man that this parasite is a serious one in any animal and that conditions somewhat similar to those in man may be present in swine. It has been reported that as a rule only 3 to 12 of these flukes are found in a pig under natural conditions.

Treatment.—Beta naphthol has been largely used in China for the removal of these worms, and is undoubtedly rather effective in removing them, as the results obtained by Barlow show. Barlow recommends that it be used in repeated small doses. Sweet gives 3 doses of 10 to 15 grains each at half-hour intervals, followed by Epsom salts in 2 hours. It would seem probable that both male fern and kamala would also be effective in removing this worm. The dose of oleoresin of male fern for swine is 1 to 2 drams; the dose of kamala would probably be about 1 to 4 drams. The single dose of beta naphthol for swine would be about 20 to 30 grains for animals of average size.

Prophylaxis.—The measures recommended for the control of *Fasciola hepatica* (See page 9) would be of value in the case of this fluke.

FASCIOLOPSIS RATHOUISI (Poirier, 1887) Ward, 1903

Rathouis's fluke

Synonyms.—*Distomum rathouisi* Poirier, 1887. Ward regards this as a good species; Odhner regards it as *F. buski* (See page 9).

Hosts.—Primary: Swine and man; secondary: Unknown.

Location.—Intestine and bile ducts.

Morphology.—*Fasciolopsis*: 1.5 to 1.9 cm. long by 8.5 mm. to 1.05 cm. wide by about 3 mm. thick. Cuticle spiny. Bluntly oval or eliptical (Fig. 4), with a short cephalic cone which is absent in *F. buski.* Oral sucker subterminal and about twice its diameter distant from the ventral sucker. Esophagus very short. Cirrus pouch convoluted. Testes in same median longitudinal field (Poirier says the same transverse zone), more compactly branched, wider and denser than in *F. buski.* Ovary on right side, small and coarsely branched. Uterus in broad, closely grouped coils. Eggs 150μ long by 80μ wide, thin-shelled.

Fig. 4. *Fasciolopsis rathouisi.* Entire worm. Ventral view. Enlarged. From Stephens, 1916, after Claus.

Life history.—Presumably similar to that of *F. buski*; as noted above, these two flukes may be identical.

Distribution.—Asia (China).

Pathology.—Similar to that of *F. buski* (See page 11). It is said to cause diarrhea, emaciation and occasionally jaundice.

Treatment and Prophylaxis.—See *F. buski*, page 11.

PARAGONIMUS WESTERMANI (Kerbert, 1878, emend. Kerbert, 1881) Looss, 1905

The lung fluke of swine

Synonyms.—*Distomum westermanii* Kerbert, 1878, of Stiles and Hassall, 1900; *Paragonimus westermanii* (Kerbert, 1878) Stiles and Hassall, 1900; *Paragonimus kellicotti* Ward, 1908; *Paragonimus ringeri* (Cobbold,

1880) Ward and Hirsch, 1915. Ward and Hirsch regard the species occurring in swine as different from that in man and that in the tiger, differences in the cuticular spines being regarded as of specific value. Japanese workers, including Kobayashi, have reached the conclusion that the forms from man, dog, cat and swine are identical. Vevers concurs with Ward and Hirsch in regarding the fluke from the tiger as *P. westermani,* that from man as *P. ringeri*, and that from dog, cat and swine as *P. kellicotti.*

Hosts.—Primary: Swine, dog, cat and man; secondary: Crabs or other crustaceans, and snails. In Japan this lung fluke has as second intermediate hosts fresh-water crabs (*Potamon* (*Geothelphusa*) *obtusipes, P.* (*Ge.*) *dehaanii, Sesarma dehaanii, Eriocheir japonicus* and *Pseudothelphusa iturbei*), and as first intermediate hosts snails of the genus *Melania* (*M. libertina,* according to Ando, and also *M. paucicincta, M. extensa, M. nodiperda* and *M. nodiperda* var. *quinaria,* according to Kobayashi) and also *Ampullaria luteostoma,* according to Iturbe. Kobayashi thinks the crayfish, *Astacus* (*Cambaroides*) *similis,* may act as a host, and Ogi has found cysts, thought to belong to this species, in *Astacus japonicus.*

Location.—In the lung tissue, occasionally elsewhere, in primary host; in muscles and hypodermis, usually, and liver, occasionally, in crustacea; and in the liver of snails. This fluke has been reported from under the peritoneum, in the liver, spleen, heart, sternal region, cheek and brain of primary host. According to Nakagawa, the cercariae from the gills of crayfish are those of *Stephanolecithus parvus,* developing in the dog, and those in the liver may belong to *Distoma kalapai,* developing in the dog and cat.

Morphology.—*Paragonimus*: Thick, oval to elongate-piriform flukes, the anterior half of the body being thicker, as a rule, than the posterior half (Fig. 5). According to Ward and Hirsch the swine lung-fluke is more slender and pointed than the lung-fluke from man. A cross-section of the worm is nearly circular, the ventral surface tending to flatten. The flukes are 3 to 14 mm. long by 2 to 6 mm. wide, and 3.5 to 5 mm. thick. The oral and ventral suckers and the excretory pore are prominently visible; the genital pore is usually indistinct. Oral sucker terminal or ventro-subterminal, surrounded by a narrow white zone free from vitelline glands; sucker 750μ in diameter. Ventral sucker 800μ in diameter and somewhat in front of the middle of the body. Intestinal ceca convoluted. Genital pore median, behind the ventral sucker. The ovary is posterior and to the left of the ventral sucker and is lobate. The testes are side by side in the posterior portion of the body and are irregularly lobed. The vitellaria are extensive,

leaving only a median dorsal and ventral space free. Laurer's canal present. Eggs (Fig. 6) 78 to 96μ long by 48 to 60μ wide, the end opposite the operculum tapering pointedly.

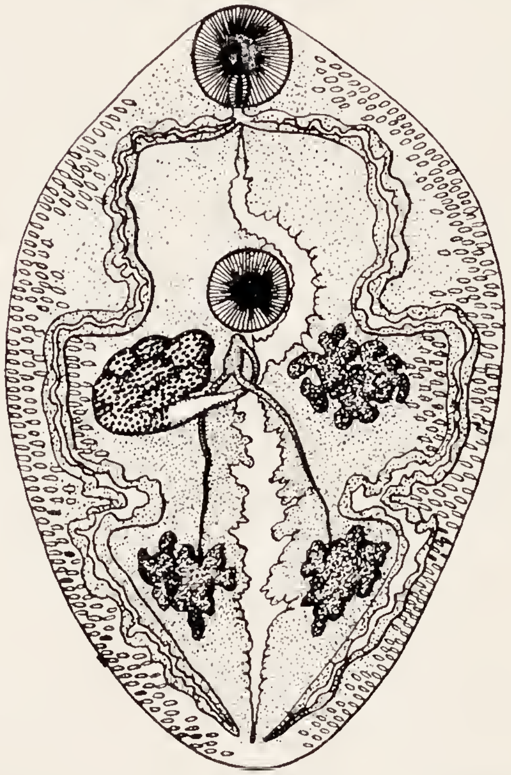


Fig. 5. *Paragonimus westermani.* Entire worm. Ventral view. Enlarged. From Stephens, 1916, after Leuckart.

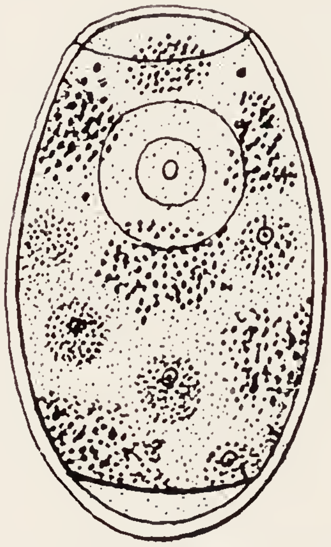


Fig. 6. *Paragonimus westermani* (*P. ringeri*). Egg from sputum, showing ovarian cell, vitelline cells and granules. x 1000. From Stephens, 1916, after Katsurada.

Ward and Hirsch and also Vevers report that in *P. ringeri* spines are arranged in clusters, whereas in *P. westermani* and in *P. kellicotti* they are single and not grouped. In *P. westermani* each spine consists of 2 fused spines, while in *P. kellicotti* each spine consists of 4 or more spines fused to form a chisel-shaped spine with a serrate edge.

Life history.—This has been investigated by Nakagawa, Yokogawa, Kobayashi, Yoshida, Iturbe and others. The miracidia develop in about 16 days under the most favorable conditions. They then enter certain species of snails and develop to cercariae. These then attack certain species of fresh-water crabs and encyst in the gills, muscles and hypodermis of the body and all appendages, and in the liver. The process of encystment requires about 30 days, and according to Kobayashi, flukes may remain alive in the crab for at least 6 years. Cysts may be released from injured crabs and occur free in water and may live for 2 or 3 weeks. It is, therefore, probable that infection may take place by eating crabs or other infected crustacea uncooked or by drinking water containing the live cysts. When these encysted flukes are

swallowed, they escape from their cysts into the stomach or intestine, pierce the wall of the digestive tract and wander about for a time in the abdominal cavity, then pierce the diaphragm and enter the thoracic cavity, where they wander about for a time, and finally enter the lungs by way of the pulmonary pleura. Erratic specimens may wander to the brain, spinal canal, liver, mesenteries, peritoneum, and other sites. Having reached their destination in the lungs the flukes become mature and the eggs which are formed usually escape to some connecting bronchiole and are coughed up in the sputum and commonly swallowed, escaping to the exterior in the sputum or feces, thus completing the cycle.

Distribution.—United States, Japan, Korea, Venezuela, Yucatan, and Peru. The fluke was found for a period in 1898 in 1 per cent of the swine slaughtered at the Cincinnati abattoir, according to Stiles and Hassall. It is found occasionally in meat inspection at various points. It has been reported from swine in Japan. It is assumed here that this is the same species as the American form, but it is recognized that this question is debatable.

According to Vevers, *P. kellicotti* occurs in North and South America and Asia, *P. ringeri* is confined to China and Japan, and *P. westermani* occurs in Asia (India and the Malay States).

Pathology.—This fluke occurs in cysts in the lungs of swine, one or more being present in a cyst, the flukes and their eggs setting up a localized inflammatory reaction. From the exterior view superficial cysts show as dark areas (Fig. 7), or if deeper may cause a superficial swelling on the lung surface. In man, similar infections give rise to spitting of blood, known as parasitic hemoptysis. Since the pig does not spit, the presence of blood in the saliva is apt to go unnoticed and no description of the clinical entity resulting from infestation with this worm in swine is known to us. The condition is usually only diagnosed post mortem. Ando and Yamata find that heavy infestations in man can be diagnosed by the use of x-rays, but light infestations resemble those with tubercle. Ando has reported a positive complement fixation reaction in man and dogs. In man, aberrant flukes wandering to the brain give rise to a parasitic form of Jacksonian epilepsy, and it is possible that something of the sort occurs at times in swine, though we have no information on this point. If present in large numbers the invading larvae may cause serious lesions or even death. Ando reports that existing infestations confer partial immunity to subsequent infestations.

Treatment.—No treatment has been attempted in swine to our knowledge. In man it is said that no fatal case of pulmonary hemorrhage from infection with this worm is known, and it has been found that patients who are removed from sources of infection commonly make a good recovery as the flukes in the lungs die and the lesions heal. Ando, Kikuiko and Imamura report that emetine is beneficial in human infestations, lessening the sexual activity of the worms. Murashima reports that intravenous injections of emetine hydrochloride in one case resulted in a diminution of expectoration and the disappear-

Fig. 7. *Paragonimus westermani (P. kellicotti).* Lung of swine showing infestation. Reduced. From Stiles and Hassall, 1900.

ance of hemoptysis. Changes in the eggs were noticeable. Japanese workers have reported favorably on tartar emetic intravenously but Maxwell has not found this beneficial. Manson gave inhalations of tincture and infusion of quassia, infusion of kousso, solutions of tur-

pentine and of santonin in spirits of wine. Fumes of burning sulphur were inhaled twice daily for a week by 1 patient and for 2 weeks by another; in one of these cases, the patient regarded himself as cured, since he stopped coughing and spitting blood. In general, rest, tonic and stimulant treatment and good food have been regarded as the indicated treatment, with removal from infected districts when feasible. The dead parasites become disintegrated and are apparently coughed up in some cases.

Prophylaxis.—Crustacea should be eaten only when cooked and only pure drinking water should be used. Where swine may feed on crabs, crayfish and other crustacea, means may be taken to fence them away from such areas as are inhabited by these animals. Eradicative measures are indicated against crayfish on other grounds, as well as in their capacity as possible carriers of these flukes. Measures for controlling snails are also indicated. The use of copper sulphate to make a solution of 1:1,000,000 has been suggested for this purpose by Chandler.

OPISTHORCHIS FELINEUS (Rivolta, 1884)

Blanchard, 1895

The cat liver fluke

Synonyms.—See Parasites of Dogs.

Hosts.—Primary: Swine and dog, cat, etc.; secondary: Cyprinoid fish and snails (See Parasites of Dogs). Normally parasitic in carnivores; developed in pig by feeding experiment.

Location.—Gall ducts of primary host; in muscles of fish.

Morphology.—See Parasites of Dogs. These flukes are 7 to 18 mm. long by 1.5 to 2.5 mm. wide, yellowish-red and almost transparent when fresh.

Life history.—See Parasites of Dogs. Ciurea has developed this species in the pig by feeding encysted larvae in the fish, *Tinca tinca*.

Distribution.—See Parasites of Dogs. Not reported from the United States.

Pathology, treatment, etc.—See Parasites of Dogs.

MICROTREMA TRUNCATUM Kobayashi, 1920

The truncated fluke of swine

Hosts.—Primary: Swine; secondary: Unknown, but probably snails or other invertebrates.

Location.—Liver of primary host.

Morphology.—*Microtrema*: Flukes 12 to 13 mm. long by 5 to 6 mm. wide and 1.5 to 2 mm. thick. Body somewhat tongue-shaped (Fig. 8), postero-lateral margins parallel, body abruptly narrowed anteriorly and truncated posteriorly. Oral sucker somewhat ventral, usually elongated transversely, and 600μ wide. Ventral sucker slightly posterior to equator of body, 7 to 7.5 mm. from anterior end, round or transversely elongated, 300 to 400μ wide. Genital aperture in median line, 1 mm. anterior to ventral sucker. Alcoholic specimens pale to brown, with uterus and vitellaria visible. Cuticle armed with spines, except postero-ventrally; spines often in groups of 3 to 4, arranged in transverse or diagonal rows. Short prepharynx, pharynx 500μ long by 400μ wide, esophagus slightly shorter than pharynx, intestinal ceca parallel to lateral margins of body until near posterior termination, then turning

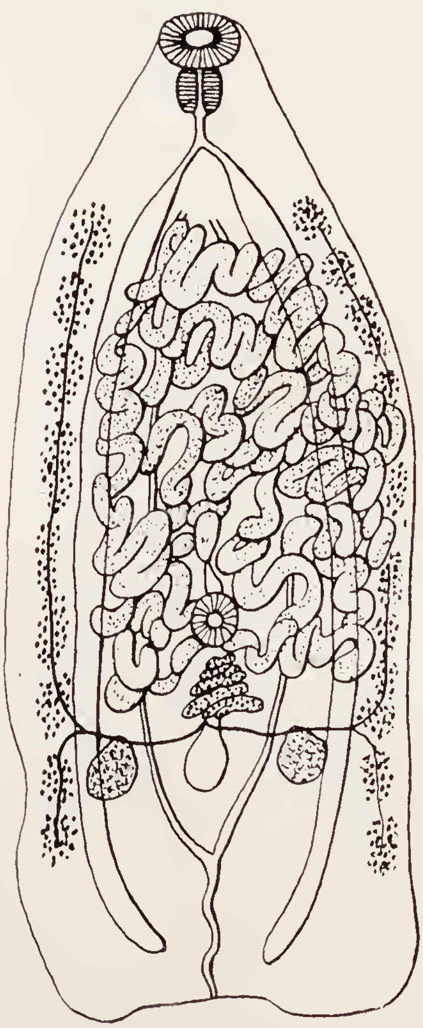


Fig. 8. *Microtrema truncatum*. Entire worm. Ventral view. Enlarged. From Kobayashi, 1920.

slightly toward median line. Excretory vesicle Y-shaped, the stem branching near posterior end of receptaculum seminis; excretory pore terminal. Testes in same transverse plane near the corresponding

intestinal branch in posterior end of body; shape irregular and slightly lobate. Ovary in median line between testes or slightly anterior, broadly triangular and composed of numerous lobes. Receptaculum seminis ellipsoid, posterior to ovary. Laurer's canal ends blind. Vitellaria external to intestinal ceca and extending from anterior extremity of ceca to level of receptaculum seminis. Uterine loops occupy entire region between ceca anterior to testes and ovary. Eggs 26 to 33μ long by 13 to 16μ wide, with distinct operculum; those in distal part of uterus contain well developed miracidia.

Life history.—Unknown; probably involves intermediate stages in snails or other invertebrates.

Distribution.—Formosa.

Pathology.—Unknown.

Treatment.—Unknown.

Prophylaxis.—Unknown. Care in regard to uncooked food and avoidance of impure or contaminated drinking water are indicated.

CLONORCHIS SINENSIS (Cobbold, 1875) Looss, 1907

The Chinese liver fluke

Synonyms.—*Distomum sinense* Cobbold, 1875; *Distomum endemicum* Baelz, 1883; *Clonorchis endemicus* (Baelz, 1875) Looss, 1907. Kobayashi regards *Cl. endemicus* as a synonym of *Cl. sinensis.*

Hosts.—Primary: Swine, man, dog and cat; secondary: Snails (*Bythinia striatula* var. *japonica*, according to Muto; *Melania libertina* may be a host, Nakagawa surmises) and cyprinoid fish (*Pseudorasbora parva, Leucogobio guentheri, L. mayedae, Sarcocheilichthys variegatus, Pseudoperilampus typus, Parachcilognathus rhombeum, Acheilognathus lanceolatum, A. lambatum, A. cyanostigma, Abbotina psegma, Biwia zezera, Carassius auratus*).

Location.—In the biliary canals of the primary host (swine); in the muscles and subcutaneous tissue of the secondary host (fish).

Morphology.—*Clonorchis*: Narrow, elongate flukes (Fig. 9), 10 to 19 mm. long by 2 to 4 mm. wide, attenuated anteriorly and rounded posteriorly, reddish or almost transparent. The cuticle is smooth. Anterior sucker larger than the posterior, the latter located in the anterior fourth of the body. The testes are very much branched and located one behind the other, in the posterior fourth of the body. The ovary is lobed and is in the median line anterior to the testes. The vitellaria are in the middle and anterior thirds of the body and extend about one-third of the body length. Genital pore just anterior to

acetabulum. Uterus extends in coils from ovary to genital pore. No cirrus or cirrus pouch. Eggs (Fig. 10) 29 to 30μ long by 16 to 17μ wide, containing a miracidium while in utero.

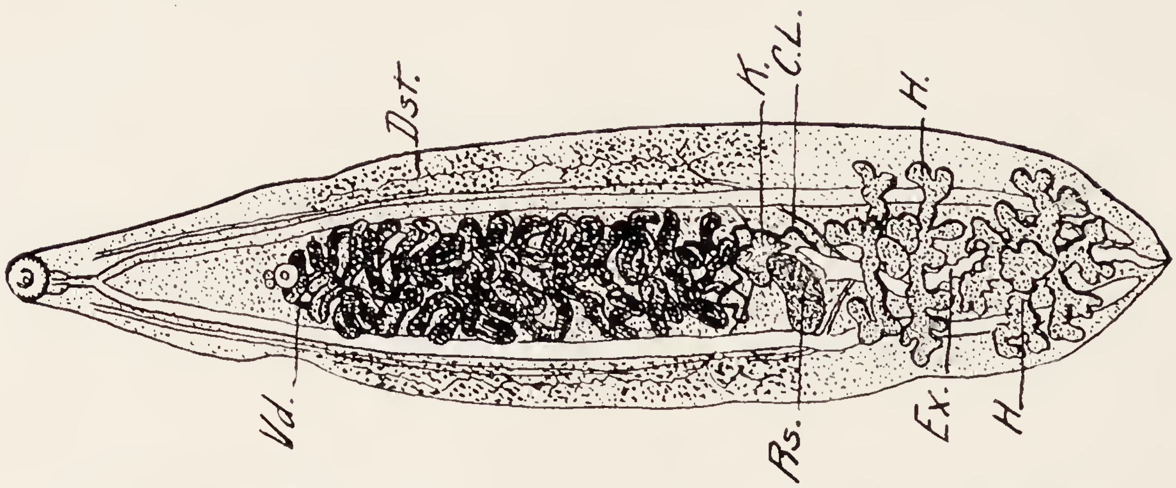


Fig. 9. *Clonorchis sinensis.* Entire worm. Ventral view. x 4½. From Stephens, 1916, after Looss, *vd,* vas deferens, terminal part; *dst,* vitellaria; *k,* ovary; *rs,* receptaculum seminis; *cl,* Laurer's canal; *ex,* excretory bladder; *h,* testes.

Life history.—The eggs pass out in the feces and the miracidia may escape from the egg in water and enter suitable species of snails. There is also the possibility that the eggs may be eaten by snails. In any event the miracidia develop to sporocysts in 3 weeks, these giving rise to cercariae, the stage infective for cyprinoid fish. When the fish eat the infected snails, the larval flukes develop to encysted forms in the muscles and subcutaneous tissue of the fish. These forms were

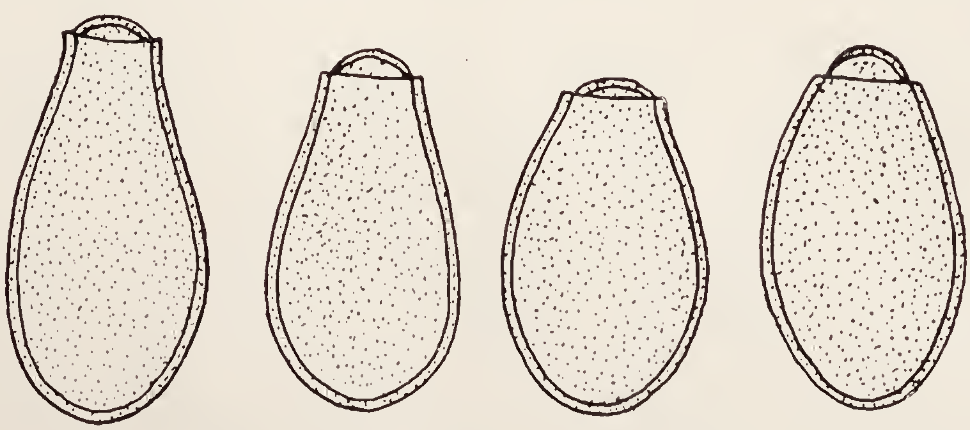


Fig. 10. *Clonorchis sinensis.* Eggs. x 900. From Stephens, 1916, after Looss. The knobs on the eggs are not shown.

found by Kobayashi, who fed them to experiment animals. When these infested fish are eaten by swine or other suitable mammalian hosts, the young flukes are found in the bile ducts and gall bladder in 15 hours, or as early as 6 hours, according to Mukoyama, indicating that they probably enter the liver by way of the common gall duct,

instead of penetrating the walls of the digestive tract and entering the liver through the capsule of Glisson, as in the case of the common liver fluke of sheep.

Muto has found experimentally that this fluke may live from 3.5 years to over 4 years in the dog, the worms then showing changes which are perhaps indications of senility.

Distribution.—China, Japan and Batavia. Querens has reported a case from man in Cuba that may have been acquired there.

Pathology.—In man, these flukes produce a condition of aqueous cachexia, with edema, exudates and development of "pot belly." The symptoms in swine, though not so well described or studied, are presumably somewhat similar. Massive infestations in man are not uncommon, and in 1 case reported by Sambuc and Beaujean, about 21,000 flukes were found in the patient at autopsy and a large number of others present in this patient were lost and not counted. Querens reports a case with uncontrollable vomiting followed by death. Toide studied 38 cases and finds that anemia is rare and not pronounced, leucocytosis is never met with, eosinophilia, never very marked, occurred in 24.3 per cent of his cases, pathological red or white blood cells were not found, and no relation was observed between the number of eggs, the blood picture and the severity of the symptoms.

Treatment.—Darling, Barber and Hacker state that they found oil of chenopodium successful in the treatment of persons infested with *Clonorchis*. As they were treating patients for hookworm, presumably the dose used for hookworm in man would be successful against these flukes in man. The indicated dose of chenopodium for experimental use in swine against these flukes would be that used for the removal of ascarids, i. e., about 1 mil for every 25 pounds of live weight of animal, up to a maximum dose of 8 mils, the dose to be accompanied by 1 ounce of castor oil for pigs weighing 50 pounds or under, by 2 ounces for animals weighing 50 to 100 pounds, and by 3 to 4 ounces for animals weighing much over 100. In man the intravenous injection of 0.4 gm. of diarsenol has been reported as causing immediate improvement in a patient. Brug reports favorable results in a human case from intravenous injections of a total of 2.95 gm. of tartar emetic in amounts of 10 to 120 mg. in 4 series of daily treatments, with periods of rest between treatments, but no flukes were found in the feces. Shattuck has reported 2 apparent cures in 3 cases.

Prophylaxis.—This would consist in such measures as would prevent swine from eating uncooked fish that could serve as carriers of the infection. Well cooked fish would be safe, as Kobayashi found

that temperatures of about 100°C would kill the larvae. Muto has suggested that water supplies be filtered to remove encysted larvae of this fluke. This precaution seems to be based on the presence of larvae present in water under conditions not accounted for in available literature on the life history.

METORCHIS ALBIDUS (Braun, 1893) Looss, 1899

The white liver fluke

Synonyms.—See Parasites of Dogs.

Hosts.—Primary: Swine, cat and dog; secondary: Fish, according to Askanazy. Normally parasitic in carnivores; developed in swine by feeding encysted flukes in fish, *Tinca tinca*, by Ciurea.

Location.—Biliary canals of primary host; in tissues of fish.

Morphology.—See Parasites of Dogs. These are small white flukes, 2.5 to 4 mm. long by 1 to 2 mm. wide.

Life history.—See Parasites in Dogs. This involves intermediate stages in fish and probably in snails.

Distribution.—Germany, Italy and Roumania.

Pathology.—See Parasites of Dogs. This fluke appears to cause irritation and thickening of the walls of the biliary canals and later a hepatic cirrhosis.

Treatment.—Unknown.

Prophylaxis.—The avoidance of the use of raw or inadequately cooked fish as an article of diet for swine, dogs and cats is an indicated control measure for this parasite.

LOXOTREMA ROMANICUM (Ciurea, 1915) Hall, 1924

The Roumanian heterophyid

Synonyms.—*Loossia romanica* Ciurea, 1915.

Hosts.—Primary: Swine and other animals (See Parasites of Dogs); secondary: Fish and snails (See Parasites of Dogs).

Location.—Small intestine of primary host; encysted in tissues of secondary hosts.

Morphology.—See Parasites of Dogs. These flukes are from 350μ to 2.5 mm. long by 240 to 730μ wide.

Life history.—See Parasites of Dogs. This involves intermediate stages in fish and in snails.

Distribution.—Roumania and Corsica.

Pathology.—Only reported from swine following experimental feeding.

Treatment.—Thymol and naphthaline have been used for *L. ovatum*. Carbon tetrachlorid, male fern, beta-naphthol, and various other drugs might be effective.

Prophylaxis.—The avoidance of raw or inadequately cooked fish as food for swine and other animals.

EUPARYPHIUM SUINUM Ciurea, 1921

The swine echinostome

Hosts.—Primary: Swine; secondary: Apparently fish (pike, tench and bream).

Location.—Small intestine of primary host; probably in tissues of fish.

Morphology.—*Euparyphium*: Echinostomes 2.73 to 3.59 mm. long by 820μ to 1.22 mm. wide and 300 to 850μ thick, the body tongue-shaped (Fig. 11). Cuticle spiny, the spines longer than wide and rounded on their posterior ends; smaller and scarcer in the median line over the bifurcation of the esophagus. Adoral disk 420 to 530μ wide

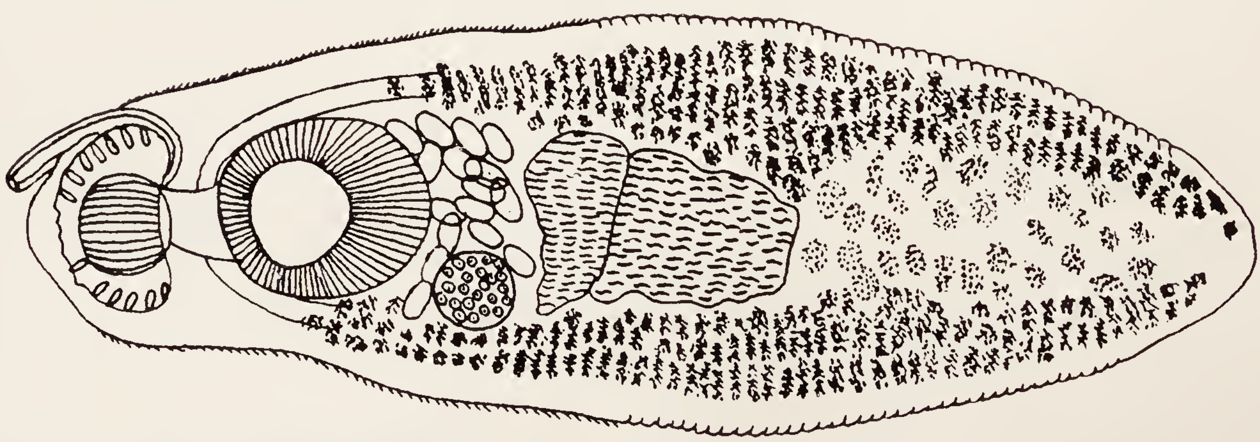


Fig. 11. *Euparyphium suinum.* Entire worm. Ventral view. x 52. From Ciurea, 1921.

transversely, incised on its ventral surface to form 2 lateral lobes united by a fold; a double row of 27 spines uninterrupted in the median dorsal line, are inserted on the margin of the disk; the spines are 68 to 99μ long. Oral sucker 300μ in transverse diameter. Ventral sucker 820μ from the anterior extremity; it is 500μ in diameter. Testes equatorial and median, one behind the other, somewhat rectangular in outline; the anterior is 290 to 490μ in diameter and the posterior 630 by 440μ. Ovary in front of and to the right of the anterior testis;

240μ in diameter. Vitellaria lateral and extending from the posterior end of the body to the region of the ventral sucker; the right and left vitellaria may terminate anteriorly at slightly different levels. Eggs lemon-shaped (citron), 117 to 127μ long by 78 to 88μ wide. Cirrus pouch extends almost to the posterior margin of the ventral sucker and contains a seminal vesicle extending its entire length; very muscular cirrus armed with small spines.

Life history.—As flukes of this species were found by Ciurea in swine fed with fish, it appears probable that the fish are intermediate hosts of the larval flukes. The life history is probably similar to that of echinostomes in general, involving larval stages in snails and subsequently in fish.

Distribution.—Roumania.

Pathology.—Unknown.

Treatment.—Unknown.

Prophylaxis.—If fish are the intermediate hosts, the avoidance of uncooked or inadequately cooked fish as food for swine and other animals is an evident prophylactic measure.

ECHINOCHASMUS PERFOLIATUS (v. Ratz, 1908) Dietz, 1909

The perfoliate fluke

Synonyms.—See Parasites of Dogs.

Hosts.—Primary: Swine, dog and cat; secondary: Fish. Ciurea has reported this fluke from a pig fed on fish, *Carassius carassius*.

Location.—Intestine of primary host; tissues of secondary host.

Morphology.—See Parasites of Dogs. These are slender flukes, 2 to 4 mm. long by 390μ to 1 mm. wide.

Life history.—Apparently involves intermediate stages in fish, and probably in snails also.

Distribution.—Roumania, Italy, Hungary and Russia.

Pathology.—Unknown.

Treatment.—Unknown.

Prophylaxis.—Avoid feeding uncooked fish.

DICROCOELIUM DENDRITICUM (Rudolphi, 1819) Looss, 1899

The lancet fluke

Synonyms.—See Parasites of Sheep.

Hosts.—Primary: Swine, sheep and other animals; secondary: Snails (See Parasites of Sheep).

Location.—In the biliary canals and gall ducts.

Morphology.—See Parasites of Sheep. These flukes (Fig. 12) are 4 to 12 mm. long by 1.5 to 2.4 mm. wide.

Fig. 12. *Dicrocoelium dendriticum.* Entire worm. Natural size. From Stiles, 1898.

Life history.—Involves intermediate stages in snails.

Distribution.—Europe, Siberia, Turkestan, Egypt, Algeria, Australia and South America. Not known to occur in the United States. See Parasites of Sheep.

Pathology.—See Parasites of Sheep. Causes catarrhal affection of gall ducts, and occasionally kills heavily infested sheep.

Treatment.—Unknown. Kamala and male fern treatments unsuccessful.

Prophylaxis.—Similar to that for *Fasciola hepatica* (See Parasites of Sheep). Avoidance of snail-infested areas, as wet, swampy pasture, or the drainage of such areas, and measures for the destruction of snails, are indicated.

EURYTREMA PANCREATICUM (Janson, 1889) Looss, 1907

The bovine pancreatic fluke

Synonyms.—See Parasites of Cattle.

Hosts.—Primary: Swine, cattle and water buffalo; secondary: Unknown, but presumably snails of some species. Dr. Faust tells me that a species of *Eurytrema,* regarded in the Orient as *E. pancreaticum,* is common in the pancreas of swine in South China. Dr. Faust reserves judgment as to the specific identity of this worm.

Location.—In the ducts of the pancreas.

Morphology.—See Parasites of Cattle. This is a small fluke, 8 to 15 mm. long by 5 to 7 mm. wide. The species in swine is smaller, a feature that might be due to occurrence in a host other than bovines or to specific differences.

Life history.—Unknown.

Distribution.—Indo-China, Japan, Java and Brazil in bovine hosts; South China for swine.

Pathology.—See Parasites of Cattle.

Treatment and prophylaxis.—Unknown.

SCHISTOSOMA JAPONICUM (Katsurada, 1904)

Katsurada, 1905

The Japanese blood fluke

Synonyms.—See Parasites of Cattle.

Hosts.—Primary: Swine, horse, cattle, dog, cat, man, etc.; secondary: Snails (*Blanfordia formosana*=*B. nosophora*=*Hypsobia nosophora.* Milton is uncertain as to the identity of *nosophora* and *formosana*) The only reference the writer has seen in regard to the occurrence of this worm in swine is in a review of a paper by Suyemori in which it is stated that he injected the cercariae from the snail host under the skin of the pig, dog, cat, rabbit and monkey and successfully infected all these hosts with the flukes.

Location.—In portal vein and mesenteric veins of primary host; in liver of secondary host.

Morphology, life history, etc.—See Parasites of Cattle.

GASTRODISCUS AEGYPTIACUS (Cobbold, 1876)

Railliet, 1893

The Egyptian amphistome

Synonym.—*Gastrodiscus minor* Leiper, 1913. According to Maplestone, *G. minor* is identical with the horse amphistome, *G. aegyptiacus.*

Hosts—Primary: Swine; secondary: Unknown, but presumably certain molluscs, such as snails, are involved. If the synonomy noted above is correct, the horse and wart-hog are also primary hosts of this fluke.

Location.—Not stated for swine; presumably digestive tract.

Morphology.—*Gastrodiscus*: See Parasites of the Horse. The only description of *G. minor* is that by Leiper in a paper proposing the specific name and is as follows: "This small fluke resembles closely

ERRATA

On page 27, the reference to figure 13 should appear in the discussion of *Gastrodiscus aegyptiacus,* not in that of *Gastrodiscoides hominis.* The figure itself is a dorsal view, not a ventral view.

Skrjabin has recently reported that the correct name for *Choerostrongylus brevivaginatus* (See page 100) is *Choerostrongylus pudendotectus* (Wostokow, 1905) Skrjabin, 1924.

the African *G. aegyptiacus* (vel *sonsinoi*) . . . It differs, however, in a number of respects, particularly in the nearness of the genital pore to the edge of the ventral disk-like expansion." Maplestone finds the genital pore in *G. aegyptiacus* to be from 400μ to 833μ from the edge of the ventral disk-like expansion, and so suppresses *G. minor*.

Life history.—Unknown; probably involves intermediate stages in molluscs, such as snails.

Distribution.—Uganda and Nigeria.

Pathology.—Unknown.

Treatment.—Presumably thymol would be effective here, as it apparently is in the case of *Gastrodiscoides hominis*.

Prophylaxis.—Avoidance of unsafe water supplies and destruction of snails are indicated measures.

GASTRODISCOIDES HOMINIS (Lewis and McConnell, 1876) Leiper, 1913

The swine amphistome

Synonyms.—*Amphistoma hominis* Lewis and McConnell, 1876. *Amphistomum (Gastrodiscus) hominis* (Lewis and McConnell, 1876) Sonsino, 1895; *Gastrodiscus hominis* (Lewis and McConnell, 1876) Sonsino, 1896.

Hosts.—Primary: Swine, usually, man, rarely, and Napu mouse deer, once; secondary: Unknown, but presumably certain molluscs, such as snails, are involved.

Location.—In digestive tract (probably intestine) of swine, and in cecum and colon of man.

Morphology.—*Gastrodiscoides*: Worms reddish when freshly collected, 5 to 8 mm. long by 3 to 4 mm. wide posteriorly. Acetabulum small with relatively large aperture (Fig. 13). The posterior disk-like portion has incurved edges which are somewhat retracted and blunted anteriorly at its union with the small anterior cylindrical or conical portion. The disk and the ventral surface are smooth, according to Leiper, and do not have the papillae or ridges said to be present by other writers. The pharynx is provided with 2 diverticula. The intestinal ceca are straight and bifurcate above or below the level of the genital pore and terminate in the acetabular zone. The genital pore is near the middle of the anterior cylindrical or conical portion of the body and may be situated on a large fleshy papilla, the surface of which bears cuticular bosses, or in a genital atrium, the condition varying with degree of contraction, according to Maplestone. The testes lie

diagonally one in front of the other in the median line, according to Khalil, and are roughly lobed; the anterior is smaller than the posterior and is near the union of the anterior and posterior portions of the body; the posterior is just anterior to the anterior margin of the acetabulum. The ovary is between the posterior testis and the acetabulum and slightly to the right of the median line, ventral to the shell gland.

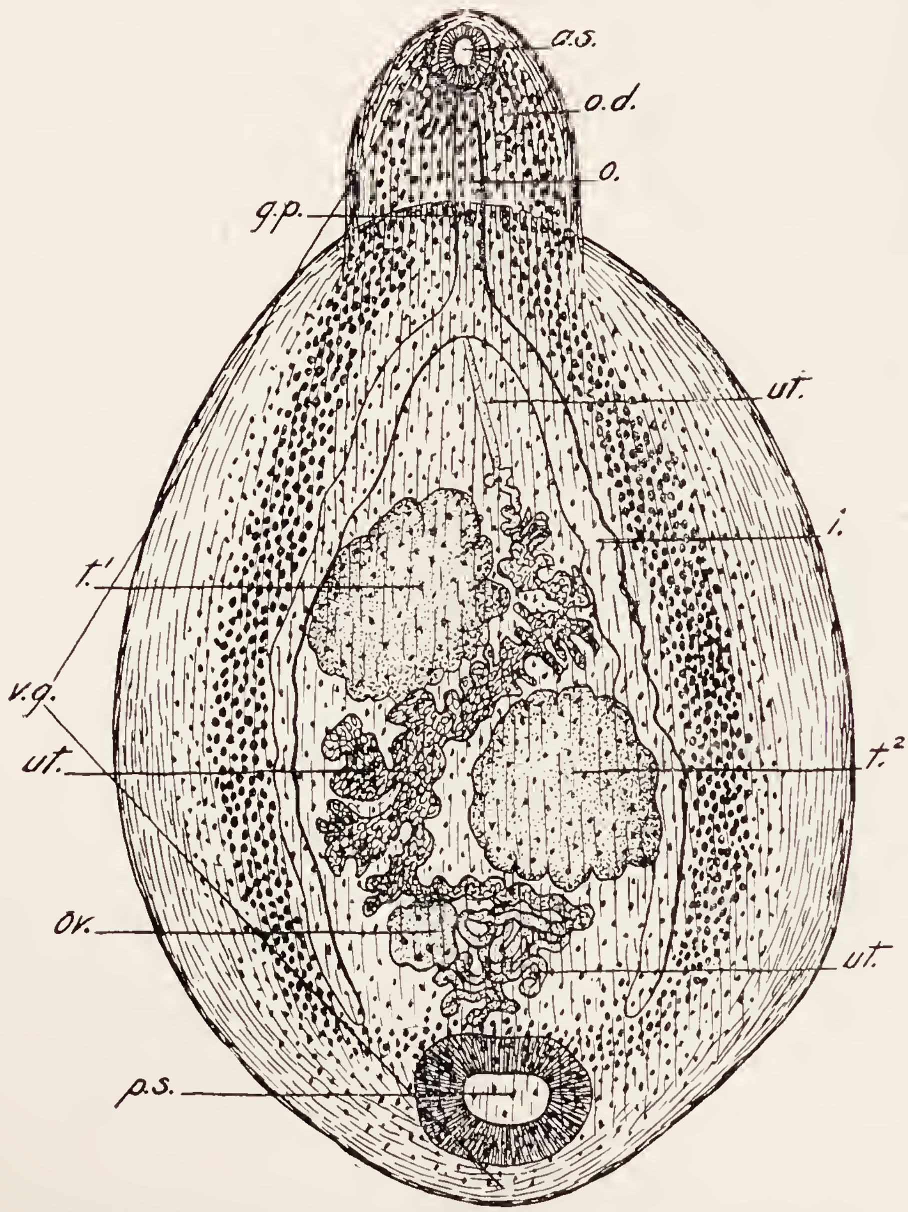


Fig. 13. *Gastrodiscus aegyptiacus.* Entire worm. Ventral view. x 6. From Maplestone, 1923. *os,* oral sucker; *od,* oral diverticulum; *o,* esophagus; *gp,* genital pore; *ut,* uterus; *i,* intestine; *t1, t2,* testes; *vg,* vitellaria; *ov,* ovary; *ps,* acetabulum.

Laurer's canal opens in front of the excretory vesicle, the latter forming an elongate sac opening posteriorly in the acetabular zone. The vitellaria are extracecal and do not extend anteriorly beyond the anterior border of the posterior testis and are most abundant between the acetabulum and the termination of the ceca. Eggs oval, 150μ long by 72μ wide.

Life history.—Unknown; probably involves intermediate stages in molluscs, such as snails.

Distribution.—Annam, Assam, Malay States, India, Cochin China, and British Guiana (in Indian immigrants). Brau and Bruyant report this parasite from 5 per cent of 100 swine at Saigon.

Pathology.—Practically nothing appears to be reported in regard to this.

Treatment.—Leiper quotes Capt. F. P. Mackie as stating that as many as 200 of these flukes were removed from one patient by the use of thymol, and as this drug appears to be effective in removing *Fasciolopsis buski* it would seem probable that it would be effective in removing these amphistomes.

Prophylaxis.—In the present state of our knowledge of related flukes, the provision of safe drinking water, avoidance of water from uncertain sources, and measures to destroy snails would be indicated.

CHAPTER II

Tapeworms Infesting Swine

DIPHYLLOBOTHRIUM MANSONI (Cobbold, 1883)

Faust and Wassell, 1921

Manson's bothriocephalid tapeworm

Synonyms.—*Sparganum mansoni* (Cobbold, 1883) Stiles and Tayler, 1902. See Parasites of Dogs.

Hosts.—Primary: Dog and cat; secondary: Swine and other hosts (See Parasites of Dogs) for plerocercoid; *Cyclops leuckartii* for procercoid.

Location.—Small intestine of primary host; subcutaneous tissue of abdomen, inguinal region, external genitalia, abdominal and thoracic walls, femur, submaxillary region, abdominal cavity and orbit of plerocercoid hosts; body cavity of procercoid host.

Morphology.—*Diphyllobothrium* (*Sparganum*): For morphology of adult worm see Parasites of Dogs. The plerocercoid larva may attain a length of 30 cm. and a width of 3 mm. to 1.2 cm. The body is ribbon-shaped (Fig. 14) and wrinkled, with the lateral borders frequently thicker than the median portion. The anterior end is usually wider and bears a retractile head provided with 2 feeble bothridia.

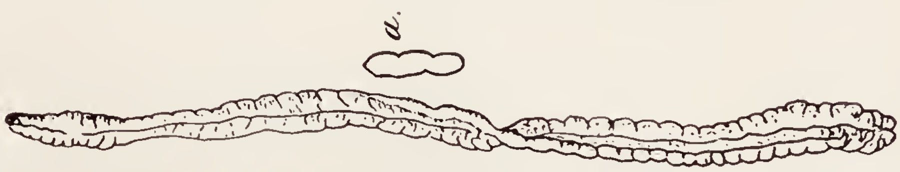


Fig. 14. *Diphyllobothrium mansoni.* Larva. *a*, cross-section. Two-thirds natural size. From Stiles, 1906, after Ijima and Murata.

Life history.—This involves intermediate stages in a copepod followed by larval stages in such intermediate hosts as swine, and the development of the adult worm in the digestive tract of the dog or cat.

Distribution.—United States, British Guiana, Malay Archipelago, Japan, French Indo-China, Annam and Australia. Reported from man in the United States (Texas) by Moore.

Pathology.—The plerocercoids may cause serious injury to the host animal, especially as a result of their wandering habits. Suppuration may occur at the site of infestation.

Treatment.—Surgical treatment is occasionally possible, but rarely practical.

Prophylaxis.—A supply of safe drinking water is essential to prevent infestation through ingestion of infested crustaceans.

DIPHYLLOBOTHRIUM RAILLIETI (v. Ratz, 1912) Meggitt, 1924

Railliet's bothriocephalid

Synonyms.—*Sparganum raillieti* v. Ratz, 1912; *Dibothriocephalus raillieti* (v. Ratz, 1912) Kotlan, 1923.

Hosts.—Primary: Dog; secondary: Swine (of plerocercoid); unknown for procercoid, but presumably entomostracans.

Location.—Small intestine of primary host; intermuscular and subcutaneous connective tissue of secondary host.

Morphology.—*Diphyllobothrium* (*Sparganum*): For morphology of adult worm see Parasites of Dogs.

Plerocercoid may attain a length of 4 to 5 or even 8 cm., contracting to 2.5 to 3 cm.; the width may be 800μ to 2.5 mm. The anterior fourth of the body may present a transverse marking suggestive of segmentation (Fig. 15) and is wider than the posterior portion; the latter is narrower, smooth, of almost uniform width and terminates in an almost rectangular truncation. The scolex appears to be very variable in form, presenting a median depression or lateral cuticular depressions simulating a taenioid sucker or other forms due to contractions and expansions. Spherical calcareous corpuscles especially abundant in anterior end of worm.

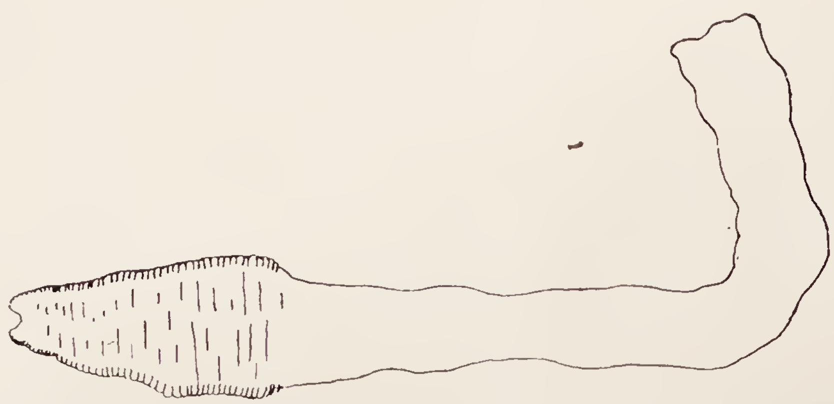


Fig. 15. *Diphyllobothrium raillieti.* Larva. x 6 2-3. From Kotlan, 1923.

Life history.—Kotlan has developed the adult worm in a dog by feeding the plerocercoid from the pig, eggs appearing in the feces in 19 days. Kotlan's attempt to infect himself by swallowing a plerocercoid failed, as did attempts to infect certain entomostracans by means of the

ciliated embryos from the egg. The development of the embryos in the egg required two and one-half months and they escaped from the shell 2 weeks later. Presumably the procercoid would have developed from these embryos in the right entomostracan host, and the procercoids would have developed to plerocercoids when swallowed in their infested hosts by pigs.

Distribution.—Hungary and probably Serbia (found in Serbian swine).

Pathology.—The site of infestation with the plerocercoids may show serous infiltration and small hemorrhages. The plerocercoids may be encysted or lie free between the muscle fibres. The surrounding tissue may show a dark brownish-red coloration.

Treatment.—In the present state of our knowledge the only remedial measure would be surgical, a quite impractical procedure, and the parasites would probably only be found post mortem as a rule.

Prophylaxis.—This would consist in providing swine with a safe water supply, so far as we can judge at present, to prevent swallowing infested crustaceans. However, definite prophylactic measures cannot be outlined until the entire life history is better known. It is evidently desirable to prevent dogs eating raw pork.

THYSANOSOMA GIARDI (Moniez, 1879) Stiles, 1893

Giard's thysanosome

Synonyms.—See Parasites of Sheep.

Hosts.—Primary: Swine, accidentally and rarely, sheep and cattle, as customary parasites; secondary: Unknown.

Kholodkowsky records specimens of this tapeworm from swine. Stiles found 3 reported cases of tapeworm in swine. Oettle has reported 1 case of late years. There are no tapeworms known to be normal parasites of swine and records of the sort are open to suspicion on the ground that the swine may have eaten sheep intestines a short time before being killed and examined, the tapeworms found having been in the sheep intestines when eaten. The food habits of swine are such that if they were capable of infestation with tapeworms they should have them commonly instead of displaying what amounts to virtually entire freedom from these worms. However, the writer has seen 1 case in which a tapeworm from swine was sent in to the Bureau of Animal Industry from Antigua, British West Indies. The genital organs were only faintly and partially developed, making even a generic diagnosis impossible. The worm was evidently an anoplocephaline

tapeworm, which seems to have developed in an unusual host and never matured. Such development has been reported for other worms. Another tapeworm from swine sent in from Antigua was apparently a species of *Moniezia* or *Cittotaenia*, and in this case also the genitalia were imperfectly developed.

Location.—Small intestine.

Morphology.—See Parasites of Sheep. These worms are 1 and 2 meters long by 5 to 6.5 mm. wide.

Life history.—Unknown.

Distribution.—Kholodkowsky's record of this tapeworm from swine is from Russia; for the distribution of the tapeworm in its normal hosts, see Parasites of Sheep. Oettle's case was in Germany; no species was named.

Pathology.—See Parasites of Sheep. Of no importance in the case of swine.

Treatment.—There is no likelihood that any treatment will ever be needed for this tapeworm in swine. For the treatment in its normal host, see Parasites of Sheep.

Prophylaxis.—Apparently the only prophylaxis necessary for preventing this worm from occurring in swine consists in preventing swine from eating worm infested intestines of sheep. The general prophylaxis for the worm in its normal host is unknown, owing to our ignorance of its life history.

Detmers in 1879 and Stiles in 1895 reported a total of 3 cases of other strobilate tapeworms said to occur in swine in the United States, but the evidence in their cases, as Stiles notes, suggests that the swine had eaten sheep or cattle intestines which were infested with tapeworms and were slaughtered in time to have these worms found in the intestine of the swine.

PARAMONIEZIA SUIS Maplestone and Southwell, 1923

The Australian swine anoplocephalid

Hosts.—Primary: Wild pig (*Sus scrofula*); secondary: Unknown. Not known from domestic pig; included here on account of rarity of tapeworms in pigs.

Location.—Intestine.

Morphology.—*Paramoniezia*: The genus and species differs from *Moniezia* in that on the left side the cirrus is sometimes dorsal and sometimes ventral to the vagina. Worm lancet-shaped, 12 cm. long

by 1 cm. wide. Head small, 300μ wide; unarmed and without rostellum; suckers extremely small. No neck. Segments always wider than long, their free edges imbricated. Mature segment 200μ long by 9 mm. wide. Genital pores double. Ventral excretory vessel large, about 150μ in diameter. Testes numerous, at least 300, extending on each side almost to lateral margin, not grouped around ovary but present clear across segment in 4 or 5 transverse rows in 1 to 3 layers. Cirrus pouch with internal vesicula seminalis; the pouch does not cross the excretory vessel. Ovary paired in each segment. Vagina always ventral to cirrus pouch on right side, but dorsal or ventral on left side. Uterus develops as a tube without outpocketings, extending to the extreme edge of the segment on each side. Eggs 45μ in diameter; the hexacanth embryo 24μ in diameter; pyriform apparatus absent.

Life history.—Unknown.

Distribution.—Townsville, North Queensland, Australia.

Pathology, treatment, etc.—Unknown.

TAENIA SOLIUM Linnaeus, 1758

Pork measles, the pork bladderworm, or the armed bladderworm

Synonyms.—*Taenia cellulosae* Gmelin, 1790; *Cysticercus cellulosae* (Gmelin, 1790) Rudolphi, 1808.

Hosts.—Primary: Man; secondary: Swine, dog, man, and, possibly, horse. Ransom has reported what may be this parasite from the horse in the United States.

Location.—In small intestine of primary host, man; in the musculature of the secondary host, as a rule, but also in the brain, eye, lungs, liver, pancreas and spleen, musculature of esophagus and stomach, and subcutaneous tissue. Railliet and Morot report this parasite from the pancreas.

Morphology.—*Taenia*: The pork measle bladderworm is ellipsoid (Fig. 16), usually 6 to 10 mm. long and 5 to 10 mm. wide, with a white spot corresponding to the invaginated scolex. The head is armed with a double row of 24 to 32 hooks, the larger 160 to 180μ long and the smaller 110 to 140μ long (Fig. 17). The head and neck are spirally disposed. The caudal bladder is mammillate on microscopic examination.

The adult tapeworm in man attains a length of 2 to 3.5 meters or more, with a maximum width of 7 to 8 mm. The head is globular, 0.6 to 1 mm. in diameter. The rostellum is short, but prominent. The hooks are similar to those described for the bladderworm. The genital

pores are irregularly alternate. Mature segments are 2.5 to 3 mm. long and 4.5 to 5 mm. wide. The ovary on the pore side of the segment is divided by the vagina as it comes in from the genital pore, a small

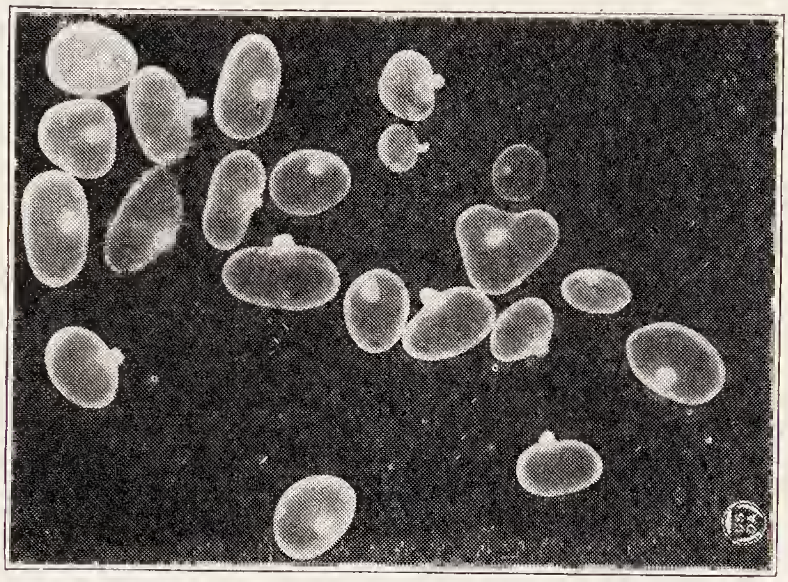


Fig. 16. *Taenia solium* (*Cysticercus cellulosae*). Larvae (bladderworms). Natural size. From Ransom, 1913.

portion of the ovary being cut off and left anterior of the vagina (Fig. 18), a feature which distinguishes these segments from those of

Fig. 17. *Taenia solium* (*Cysticercus cellulosae*). Hooks from bladderworm. Left, large hook; right, small hook. Enlarged. From Ransom, 1913.

Taeniarhynchus saginatus. Gravid segments are 10 to 12 mm. long by 5 mm. wide. The uterus has 7 to 14 thick lateral branches. The eggs (Fig. 19) are oval or almost round; the shell is thin and usually destroyed; the embryophore is thick, yellowish to dark-brown, and 31 to 36μ in diameter.

Life history.—The life history of this worm is somewhat similar to that of the unarmed tapeworm of man, *Taeniarhynchus saginatus*. The eggs produced by the adult tapeworm in the human intestine are ingested by swine when human feces are not properly disposed of. As a result of the fact that gravid segments usually pass several at a time in a short chain and also as a result of the feeding habits of swine, including coprophagy, or of the modes of feeding them, they are more likely to ingest entire segments or large numbers of eggs than cattle are to ingest segments of the beef tapeworm, and are consequently more sub-

ject to heavy infestation. In swine the embryos from these eggs make their way, probably both by transfer in the blood stream and by active migration, to their sites, usually in the musculature, and develop to bladderworms with armed heads. When these bladderworms are eaten

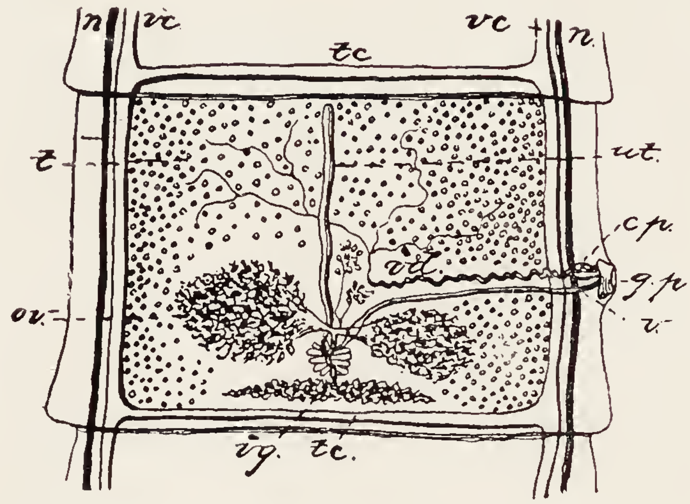


Fig. 18. *Taenia solium*. Mature segment. x 10. From Stiles, 1898, after Leuckart. *cp*, cirrus pouch; *gp*, genital pore; *n*, nerve; *ov*, ovary; *t*, testes; *tc*, transverse canal; *ut*, uterus; *v*, vagina; *vc*, ventral canal; *vd*, vas deferens; *vg*, vitellarium.

by man, they develop to tapeworms. This tapeworm is rare in man in this country, owing partly to a preference for pork products that are well cooked, in contrast to the national fondness for rare beef and rare steaks. Such infestation as occurs probably occurs as a rule from those pork products which are usually eaten raw or but slightly cooked. such as sausages, etc.

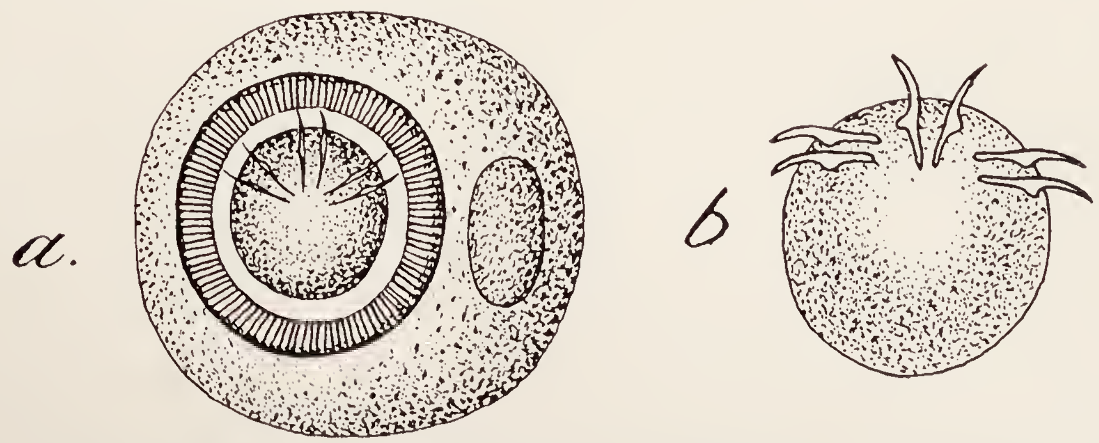


Fig. 19. *Taenia solium*. *a*, egg with shell and embryophore; *b*, onchosphere. Enlarged. From Fiebiger, 1923, after Leuckart.

According to various writers, the development of the cyst in the pig is about as follows: At the end of 9 days, an oval vesicle 33μ long by 24μ wide, with the connective tissue cyst not developed; 20 days, a transparent bladderworm, the size of a pin head, the position of the head indicated by a small point and no surrounding cyst developed;

32 days, 1 to 6 mm. long by 0.7 to 2.5 mm. wide, the position of the head indicated at equator of the cyst and the surrounding cyst thin; 40 days, head developed with hooks and suckers visible but not complete and with surrounding cyst still delicate; 60 days, size of a pea or larger, head, hooks and suckers developed, but no neck; 110 days, neck developed, transverse lines slightly visible, and head invaginated in bladder. It is usually estimated that 3 to 4 months are required for the development of the bladderworm, and Gerlach thinks that those less than 2 months old are not dangerous. The cyst may live a long time. It has caused cerebral trouble in 1 case in man for 12 to 15 years and has been visible in the eye for 20 years, though the writer is not aware whether these cysts were alive in these cases. Caseous degeneration may occur early or late It begins with the formation of cheesy spots in the capsule, followed by caseation of the bladderworm cyst and the degeneration of the suckers, the disarrangement of the hooks, and terminates in the formation of a caseous mass. It is sometimes possible to identify these degenerated worms by the finding of hooks or of recognizable fragments of capsule. There may be terminal calcareous degeneration.

Distribution.—Cosmopolitan. This tapeworm occurs in those countries where pork is eaten. The adult worm is rarely found in the United States, Hemmeter's case, reported by Stiles, being apparently the only valid record for years. More recently Moser and Emerson have each reported a case. Pork measles occurs sporadically in American swine.

Pathology.—The occurrence of pork measles in swine has been regarded by some writers as the probable cause for the Mosaic injunction prohibiting the use of pork. Aristophanes and other Greek writers refer to the disease.

The symptoms of this disease in hogs are very indefinite, according to Stiles. The parasite occurs in the muscles, especially the abdominal muscles, the muscular portion of the diaphragm, the psoas, tongue, heart, muscles of mastication, intercostals, muscles of the neck, adductors of the hind legs, and the pectorals.

In meat inspection, this parasite receives much the same attention as *Cyst. bovis*, but it is regarded as a more dangerous parasite, owing to the fact that the larval stage, or cysticercus, may occur in man in addition to the adult stage, or strobilate tapeworm, and this larval form has an unpleasant predilection for the eye and brain. Heavy infestations are not uncommon, as has been noted above, and as many as 20,000 or more cysts may occur in one carcass. Kuechenmeister has found 133 cysts in 17 grams of pork and Hall has reported a natural

infestation with an estimated 190,470 cysts in an eviscerated headless pig carcass, weighing 56 pounds. The parasite may be detected antemortem, by an examination of the under surface of the tongue in some cases, though the examination of the tongue in the live hog is not a very attractive procedure. Where heavy infestations are present, the carcass is pale and watery, decomposes easily and has a disagreeable sweet taste. When the parasites have died and undergone calcareous degeneration the condition is sometimes referred to as dry measles.

Infestation with the cysticercus in man is commonly suspected of originating in a previous infestation with the mature tapeworm in the intestine. The patient may soil the hands with feces and subsequently contaminate his food or convey the eggs to the mouth in other ways, or there is the possibility that a reverse peristalsis may carry eggs or gravid worm segments from the intestine to the stomach, with a resultant digestion of the shell and release of the embryo. Virchow reported that the proportion of these cysticerci in human cadavers in Berlin was reduced from 1 in 31 to 1 in 280, following the introduction of meat inspection.

The symptoms of cysticercosis in man vary with the location of the parasite. Swine cysticercosis is almost never diagnosed on symptoms, being only detected postmortem.

The symptoms of infestation with the armed, or pork-measle, tapeworm are much the same as for the unarmed, or beef-measle, tapeworm or for tapeworms in general. Stiles gives the general symptoms for tapeworm in man as follows: Itching at the extremities of the intestinal canal, and various dyspeptic symptoms; uncomfortable sensations in the abdomen, including the sensation similar to that experienced in a rapidly descending elevator; uneasiness, fullness or emptiness, sensations of movement attributed to the movements of the parasite, and colicky pains; disordered appetite, at times deficient and at times craving; paleness and discoloration around the eyes; fetid breath; sometimes emaciation; dull headache; buzzing in the ears; twitching of the face; dizziness; fainting, chorea, and epileptic fits. Other writers add disorders of systems other than the digestive and nervous, including even disorders of the genito-urinary, such as dysmenorrhea. The diagnosis is made on the finding of eggs or segments in the feces. In this connection we may recall that segments of *T. solium* usually pass in chains and those of *Taeniarhynchus saginatus* pass as disconnected individual segments.

Treatment.—For practical purposes, there is no treatment for cysticercosis of the musculature in swine. In man, the treatment for

this condition is surgical. The removal of the adult tapeworm from the intestine is said by some writers to be more easily accomplished than the removal of the unarmed tapeworm of man, in spite of the presence of the hooks on the head of the armed form. The treatments usually used are oleoresin of male fern, pelletierine tannate, Tanret's pelletierin, or kamala. Owing to the danger to man from cysticercosis, treatment should be given promptly and followed up when this tapeworm is found present.

Prophylaxis.—Proper disposal of human excreta is necessary to prevent infestation of swine with the bladderworm and the thorough cooking of pork and pork products is necessary to prevent infestation of man with the tapeworm.

TAENIA HYDATIGENA Pallas, 1776

The thin-necked bladderworm

Synonyms.—*Cysticercus tenuicollis* Rudolphi, 1810. See Parasites of Dogs.

Hosts.—Primary: Dog and other carnivores (See Parasites of Dogs); secondary: Swine, sheep, cattle and various ruminants.

Location.—Small intestine of primary host; imbedded in liver, or free or attached to viscera, and especially in mesenteries, in abdominal cavity of secondary host.

Morphology.—See Parasites of Cattle. This bladderworm may attain the size of a man's fist and even larger sizes have been reported. The neck is long (Fig. 20) and the head is armed with 26 to 44 hooks.

Fig. 20. *Taenia hydatigena* (*Cysticercus tenuicollis*). Larva (bladderworm). Natural size. From Stiles, 1898.

Life history—See Parasites of Cattle. When eggs from the tapeworm in the dog's intestine are swallowed by suitable secondary hosts these hosts become infested with bladderworms. Bladderworms eaten by dogs develop to adult tapeworms.

Distribution.—Cosmopolitan.

Pathology.—Cases have been reported by Leuckart, Railliet, Zschokke and Pillers where swine have died from severe infestations with this bladderworm naturally acquired, and the same effect has been produced by experimental feeding. The parasites caused peritonitis and pleurisy by the wanderings from the liver and lungs to the body cavities. Fox has reported unthriftiness and diarrhea in 8 pigs, 6 weeks old, the livers of 2 giving the appearance of varicose veins present as a result of the wanderings of the larval tapeworm. Usually no symptoms are observed in natural infestations. However, Joest refers a hepatitis interstitialis chronica of swine to this parasite. According to him, hepatophile oncospheres, of the type of *Echinococcus*, develop in the liver without wandering and cause little damage in that stage. Non-hepatophile oncospheres, destined to become bladderworms outside of the liver, wander about and cause considerable destruction and injury to tissues and capillaries. This results in chronic hepatitis. In the writer's experience the amount of chronic interstitial hepatitis in swine in proportion to the occurrence of *Cyst. tenuicollis* appears to be too great to permit of this parasite being the usual cause of this condition. Joest recognizes a diffuse hepatitis from many larvae and a multiple hepatitis from a smaller number. Areas of infiltration are found to consist almost entirely of eosinophiles. Further work may show the larvae of *Ascaris lumbricoides* to be responsible for the common occurrence of so-called parasitic livers, largely cases of chronic parasitic cirrhosis, in swine.

Treatment.—There is no medicinal treatment known for infestation with this bladderworm and in the small numbers in which it ordinarily occurs no treatment appears necessary. Surgical treatment would be highly impracticable. The condition is not diagnosed antemortem, though it is possible that a biological method of diagnosis might be devised.

Prophylaxis.—The destruction of the bladderworms found in carcasses in meat inspection is an important method of control for this parasite and it seems at present that there is a decrease in the incidence of this parasite in its adult stage in dogs in the last 15 years, probably due in part at least to increased extension of meat inspection and better abattoir methods. The deep burial or the destruction by burning or otherwise of carcasses of animals dying in the fields is another indicated measure of control. Proper supervision of the food habits of dogs and routine anthelmintic treatments to rid them of tapeworms acquired where proper supervision of their food habits is not exercised are other control measures.

MULTICEPS MULTICEPS (Leske, 1780) Hall, 1910

The gid bladderworm

Synonyms.—*Coenurus cerebralis* (Batsch, 1786) Rudolphi, 1908. See Parasites of Sheep.

Hosts.—Primary: Dog and coyote (*Canis nebracensis*); secondary: Swine, rarely, and sheep and other ungulates, usually. See Parasites of Sheep for other hosts. Ciani has reported this parasite once from swine and this record is accepted by Railliet and Mouquet.

Location.—The central nervous system.

Morphology.—The parasite in the brain may attain the size of a hen's egg. It consists of a thin bladder full of fluid and showing on its limiting membrane numerous white spots which are tapeworm heads. For details, see Parasites of Sheep.

Life history.—When a bladderworm is eaten by a dog, or other suitable host, some or all of the tapeworm heads may develop to adult tapeworms in the intestine. If the eggs passing in the feces of dogs are swallowed by a suitable secondary host this host may become infested with the coenurus or bladderworm.

Distribution.—See Parasites of Sheep. This worm occurs in the United States, Canada and Mexico, and is enzootic in Montana.

Pathology.—In sheep this worm causes striking symptoms (circling, stumbling, etc.) due to the destruction of nervous tissue by it, and unless relieved of the parasite in some way the infested animal invariably dies.

Treatment.—Surgical; treatment is a matter of no known importance in the case of swine.

Prophylaxis.—The destruction of the brains of animals dying of gid and keeping dogs free from tapeworms by adequate supervision of their food or the use of anthelmintics.

ECHINOCOCCUS GRANULOSUS (Batsch, 1786) Rudolphi, 1805

The hydatid

Synonyms.—See Parasites of Dogs.

Hosts.—Primary: Dog, cat and other carnivores; secondary: Swine and numerous other animals. In this country swine are more commonly infested with hydatids than any other of the domesticated animals and occasional consignments at abattoirs are found to have a majority of the included animals infested. In pastoral countries sheep are often the most common hosts of the larval worm.

Location.—In intestine of primary host. In practically every organ and tissue of secondary host. The liver and lungs are favored sites.

Morphology.— *Echinococcus*: The form occurring in swine is the larval tapeworm or bladderworm. This may attain the size of a child's head and has a thick laminated external wall (Fig. 21) and a delicate

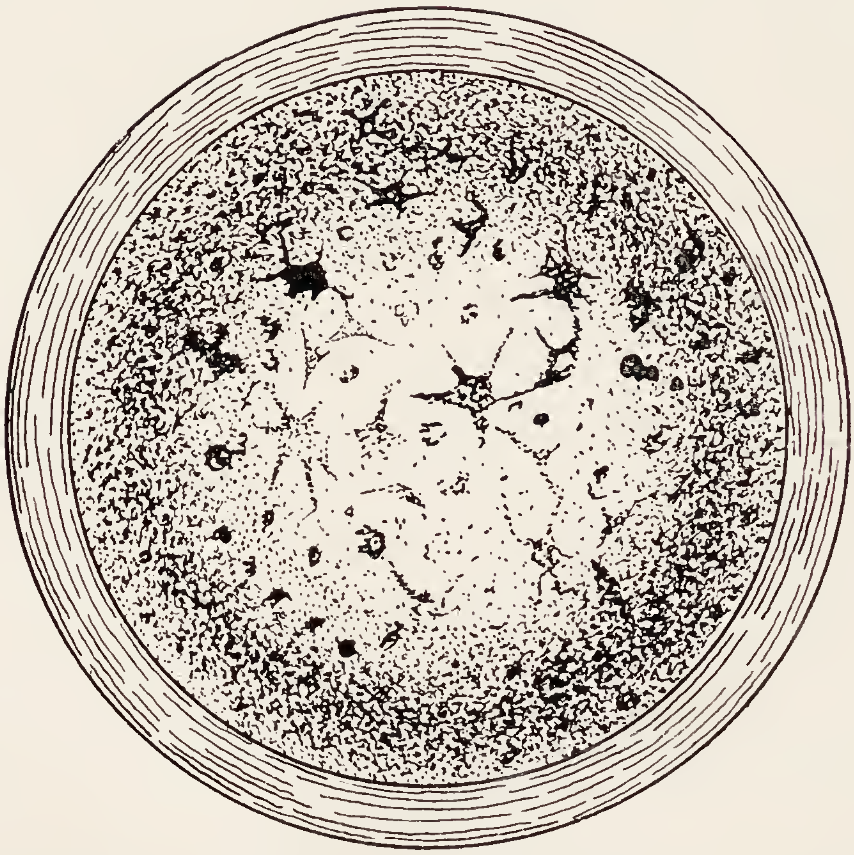


Fig. 21. *Echinococcus granulosus*. Hydatid bladder 8 weeks old. x 50. From Leuckart, 1886.

internal germinal layer. Normally this germinal layer gives rise to brood capsules, which look like so much sand to the naked eye, but which contain a number of tapeworm scolices. Up to a certain stage of its development the bladder is sterile. As a defense reaction against unfavorable conditions, the germinal layer may give rise to internal daughter bladders (rarely, according to Dévé), or the internal brood capsules may give rise to internal daughter bladders, or the scolices, by a form of degenerative evolution, may do so, while fragments of the germinal layer, the proliferative element, astray in the cyst wall, may form external daughter bladders (Fig. 22). Daughter bladders may form grand-daughter bladders, and any bladder of the series may form brood capsules containing scolices. Bladders may remain attached or become detached. Diverticula in the walls may form the racemose or multilocular forms of hyatid, sometimes resembling bunches of grapes.

Hydatids ranging downward in size from that of oranges are perhaps the commonest findings in swine, usually occurring in the liver (Fig. 23).

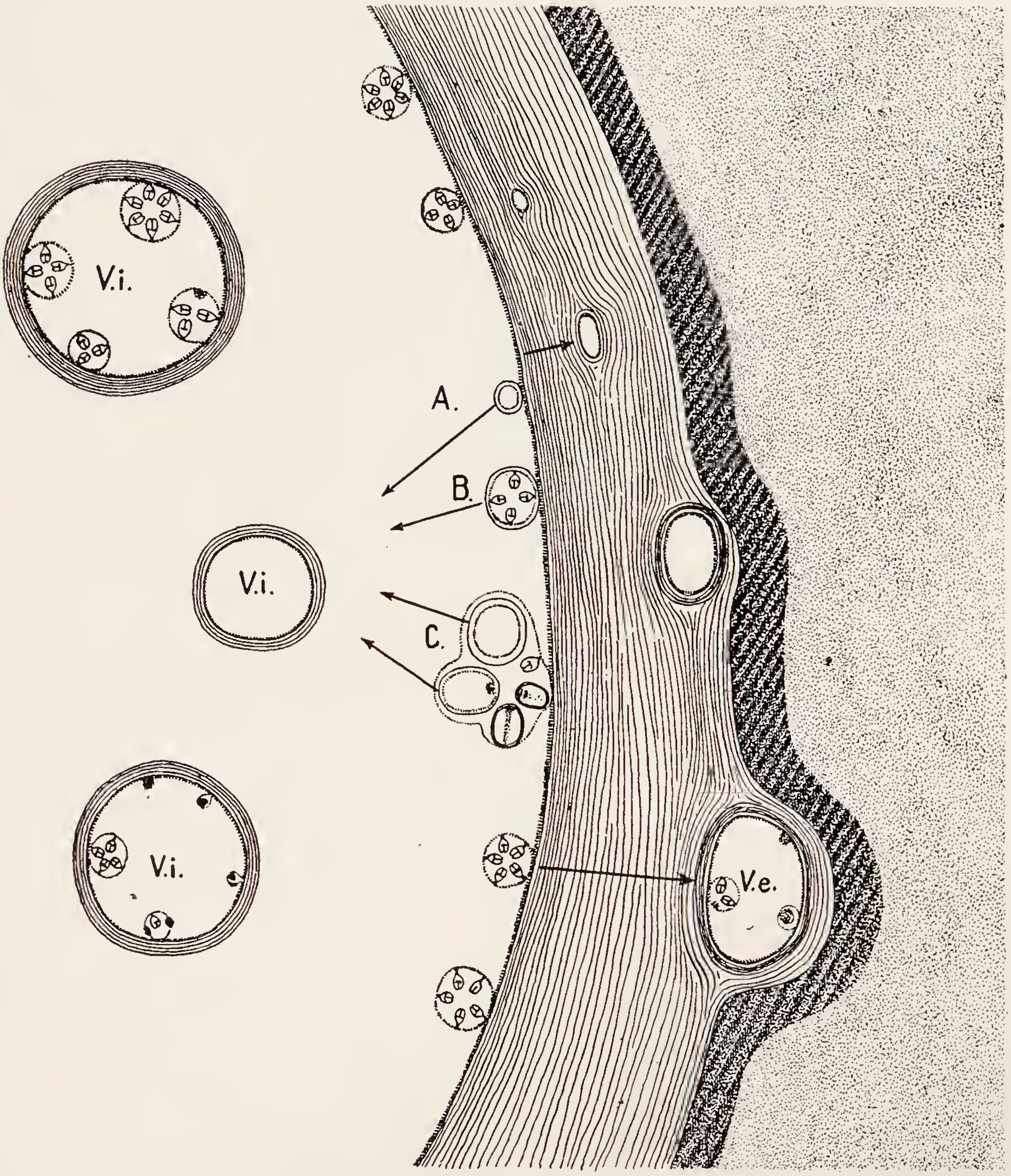


Fig. 22. *Echinococcus granulosus.* Diagram showing mode of development of internal and external daughter bladders. External daughter bladders, *V. e.*, have an intracuticular origin as shown. Internal daughter bladders, *V. i.*, have an endovesicular origin as follows: *A*, from the germinal membrane directly; *B*, from brood capsules which elaborate an external cuticle; *C*, from a scolex by vesicular metamorphosis. From Dévé, 1918.

Life history.—The eggs produced by the adult worm in the intestine of the primary host pass out in the feces and are ingested by the secondary host in contaminated food and water, as a rule. In the digestive tract the onchosphere escapes from the shell and makes its way

into the tissues. Dévé has traced the migration of the larva very closely and finds it in the portal capillaries in 3 to 5 hours; it undoubtedly goes by the portal veins. In the tissues it develops into the larval stage. During the course of development of the scolices in the brood capsules, according to Goldschmidt and to Nesvadba, the scolex may show numerous scattered spines or hooklets. When the larvae, or bladderworms, are ingested by a suitable host, the scolices develop in the intestine to form a very small tapeworm (See Parasites of Dogs).

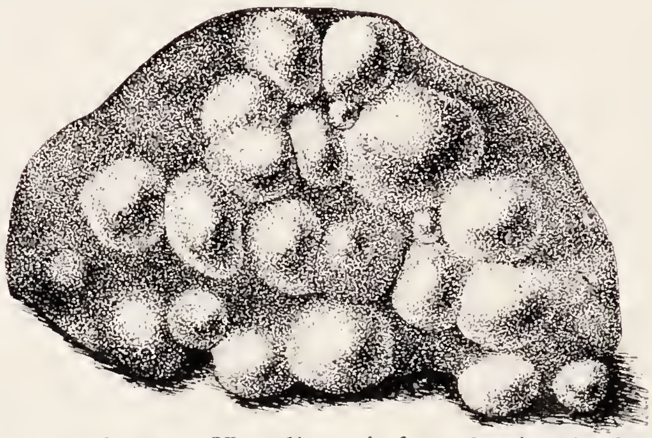


Fig. 23. *Echinococcus granulosus.* Hog liver infested with hydatids. Greatly reduced. From Stiles, 1898.

Intrauterine infestation with hydatid has been reported. Cruveilhier has reported hydatid in an infant 12 days old, and Heyfelder reports hydatid of the placenta and umbilical cord in a 7-months fetus. Dévé regards these and similar reports as erroneous. According to Leuckart, the hydatid in swine is 1 mm. in diameter in 1 month, 2 mm. in 2 months, and 1 cm. in 5 months, the heads developing some months later.

Distribution.—Cosmopolitan. In 1901 Vegas and Cranwell reported that 40 per cent of swine killed at Buenos Aires were infested with hydatid. It occurs sporadically in swine in this country. The hydatid has become a serious pest in Australia and in Brazil and is common in Siberia, Iceland and India.

Pathology.—This is a very dangerous parasite, owing to the fact that it may occur in practically any organ or tissue and may attain a relatively enormous size. The lesions due to the worm include those from pressure, resulting in pressure atrophy of organs, those from tension, due to the weight of a parasitic cyst, resulting in loss of nervous energy from pull on the mesenteries and omenta and in degrees of shock associated with such pull, and also causing associated inflammatory changes due to pressure and tension. Occasionally the hydatid will open blood vessels or other tubes. There are also indications of toxic effects, and these are especially likely to be manifest in

those cases where the parasitic cyst is invaded by bile, with partial degeneration. There are also all the numerous accidents connected with the growth of the hydatid in such locations as the brain, eye, bones, genitalia, etc. Finally, rupture of the hydatid may lead to serious or even fatal consequences, especially where the contents discharge into a blood vessel or an air passage.

Around the hydatid the host tissue commonly forms a connective tissue cyst, from which the parasite may be neatly separated as a rule. The hydatid may undergo caseous or calcareous degeneration and only be identifiable by the presence of hooks or fragments of laminated cuticle.

In hydatid infestations, the symptoms are often localized in the form of swellings, which on percussion give the characteristic vibrant response of a cyst full of fluid, with a responsive return pressure. The percussion sound is dull. Symptoms are most evident and the condition most dangerous when the hydatid occupies an important organ, or becomes very large, or becomes osseous or cartilaginous, or when there are numerous hydatids, or the hydatids rupture. No particular symptoms seem to be described for hydatid disease in swine. The symptoms vary with the site of infestation. Some extremely heavy infestations occur. In an animal killed at Bennings, D. C., the entire liver was filled with cysts about 2 mm. in diameter. In this case there was generalized icterus. It is reported that the liver of an infested pig may be increased in weight from a normal of 2 kilos to 90 kilos.

A report on the formation of hydatid antibodies in swine has been published by Weinberg. Anaphylaxis in hydatid infestation has long been known. Luridjana states that in spite of a very definite cutaneous reaction cyst puncture may not be followed by anaphylactic shock, and, conversely, in spite of a negative skin reaction, puncture may be followed by severe shock.

Treatment.—The approved treatment in man is early surgical interference. Of course, food animals that are in condition to slaughter for food may be slaughtered, the infested portions tanked or otherwise suitably disposed of, and the remainder of the carcass utilized. Hydatids that are operated on at an early stage may be removed before they have opportunity to develop daughter bladders in adjacent tissue, the complete removal later being a difficult affair. Frequently hydatids are in unoperable sites and very commonly it is unprofitable to operate. Various substitutes for operation have been devised, such as aspiration of the hydatid content or the injection of formol with a suitable type of syringe, but these are not recommended by the best authorities.

The use of oleoresin of male fern by mouth against hydatid has been tested by Dévé, but he found that it had no preventive or curative value, and Dévé and Payenneville found novarsenobenzol of no value as a prevention of infestation. In general, swine would not be operated on for hydatids, nor would the infestation be diagnosed antemortem.

Prophylaxis.—Hydatids should be carefully destroyed, so that by no possibility may they be eaten by dogs or other carnivores. This is safely accomplished in the modern abattoirs by tanking, the cysts being killed and destroyed by steam cooking under pressure, and undoubtedly modern packing house methods have done much to lessen the incidence of this disease. That it still exists is probably due to the careless methods in vogue in the small slaughter houses of the country and in cities where no interstate business is done. Here offal, especially diseased viscera, is still commonly fed to dogs, and the hydatid parasite transmitted to the dogs. In the country, farmers not uncommonly do their own slaughtering, and here again too little care is taken in the disposal of waste. Dogs should not be fed diseased viscera of any sort and should not be allowed access to slaughter houses, unless under narrowly restricted conditions which make it impossible for them to get at any portions of carcasses or to contaminate food in any way. For choice, they should be kept out entirely.

Where dogs are known to be infested with the adult hydatid tapeworm, the dog should be killed and the carcass burned or buried deep with plenty of quicklime. Such dogs are very dangerous, as an egg from the hydatid tapeworm may develop in a person with serious or fatal consequences, a thing which makes such animals entirely too dangerous to handle or to treat by anthelmintics. Too great familiarity with dogs is a common preliminary to hydatid disease in man. Stray dogs and ownerless dogs are dangerous and should be killed.

Where dogs are eaten as food in Europe, the digestive tract is excluded from use in food products, owing to the possibility of infection with this parasite.

Of the disease in man, it has been said that 50 per cent of the cases die within 5 years.

ECHINOCOCCUS ALVEOLARIS Klemm, 1883

The alveolar hydatid

Synonyms.—See Parasites of Dogs.

Hosts.—Primary: Dog; secondary: Swine, cattle, sheep and man. This parasite appears to be less common in swine than in the other

hosts listed here. Some writers are inclined to regard the larva found in man as the only true alveolar hydatid.

Location.—In small intestine of primary host; usually in liver of secondary host.

Morphology.—See Parasites of Cattle.

Life history.—Mangold fed eggs from the dog tapeworm of this species to a pig 12 weeks old. Four months later the pig was killed and 2 growths, the size of hazelnuts, found in the liver. On section these showed a sinuous multilocular structure of a hydatid. Scolices were not yet present. For other features of life history, see page 43.

Distribution, Pathology, etc.—See Parasites of Cattle.

CHAPTER III

Nematodes Infesting Swine

STRONGYLOIDES SUIS (Lutz, 1894) Linstow, 1905

The strongyloides of swine

Synonyms.—*Strongyloides longus* (Grassi, 1885) Rovelli, 1888, in part.

Hosts.—Swine. The form present in swine is referred by some writers to *S. papillosus* (Wedl, 1856) Ransom, 1911, which is a parasite of ruminants, rodents, etc. This form and the form described by Lutz may both occur in swine. See also *S. stercoralis*, page 49.

Location.—Small intestine.

Morphology.—*Strongyloides*: Parasitic generation of females only (Fig. 24). Worms 3.75 mm. long by 80μ in maximum width, according to some writers. According to Marotel, the females in the intestinal lumen are 3 to 4 mm. long by 40μ wide and are all immature, the mature females being in the mucosa where they oviposit. Very similar to *S. papillosus* from sheep and goats. Eggs 45μ long by 25μ wide, thin-shelled. Lutz says that the parasitic females are over 1 cm. long. The form found present in swine in the Eastern United States does not agree with these descriptions.

Fig. 24. *Strongyloides suis.* Parthenogenetic female, with eggs, from the intestine. Enlarged. From Fiebiger, 1912.

Free-living generation.—This consists of males and females. Lutz states that these are twice as large as *S. intestinalis* (*S. stercoralis*) from man.

Males undescribed.

Females undescribed. A specimen from swine manure examined by the author agrees quite well with *S. papillosus*. The worm was 1.18 mm. long by 60μ wide. The esophagus was 180μ long. Vulva near the middle of body, slightly post-equatorial. Only 2 eggs visible in uterus, one, well-formed, 60μ long by 30μ wide.

Life history.—Probably similar to that outlined for *S. stercoralis* (See page 49).

Distribution.—Europe, South America (Brazil) and Africa (Belgian Congo). The worms present in swine in the Eastern United States do not agree with available descriptions of *S. suis;* they may be an undescribed species, or the available descriptions may be inaccurate, or they may be *S. papillosus.*

Pathology.—According to Marotel, porcine anguillulosis may be a serious matter, the parasite causing acute generalized enteritis and sometimes proving fatal. Animals show a chronic diarrhea with colic and progressive emaciation going on to cachexia. Marotel thinks this may be due to massive infestation from constant reinfection or to the burrowing habits of the gravid female opening channels in the mucosa for secondary bacterial invasion. Diagnosis is made on the presence of the eggs, usually coupled in pairs, in the manure. Marotel's description of the pathology appears to be based on a single case. Morawetz reports cases in a sow and litter with evidence of intestinal catarrh and pronounced anemia and weakness.

Treatment.—Marotel recommends dieting, purgation and the use of such anthelmintics as male fern, thymol, areca nut and santonica. Reisinger states that he had good results from the administration of 15 to 20 grams of Levant wormseed (Santonica) three times a day, mixed with the feed, to each pig. Morawetz reports cures following the administration of a proprietary arsenobenzol product. In man good results have been reported by Stiles in some cases from the use of flowers of sulphur, and Willis has reported cures in 60 cases after repeated treatments with oil of chenopodium for *S. stercoralis* infestations.

STRONGYLOIDES STERCORALIS (Bavay, 1876)

Stiles and Hassall, 1902

The strongyloides of man

Synonyms.—See Parasites of Dogs.

Hosts.—Swine, dog, and also man.

Location.—Small intestine.

Morphology.—See Parasites of Dogs. The parasitic generation consists entirely of parthenogenetic females about 2.4 to 2.64 mm. long by 45 to 48μ wide.

Life history.—The parasitic females are said to deposit eggs in the cells lining the intestinal crypts and covering the villi. The eggs hatch and the rhabditiform larvae escape to the lumen of the intestine and

pass out in the feces. Under favorable conditions these may: (1) Develop to infective filariform larvae capable of infecting the host by way of the skin or of the mouth; or (2) develop into free-living males and females, the eggs of which develop to rhabditiform larvae and these in turn to infective filariform larvae. In the body the worms enter the blood stream and make their way in time to the lungs, where they enter the air spaces, ascend the trachea, and are swallowed, developing to adult female worms in the intestine.

Ackert and Payne report infestation with this worm in swine by feeding experiments in which infective larvae from a case of human infestation were fed with the subsequent appearance of strongyloid larvae in increasingly large numbers in the manure of the swine.

Distribution.—See Parasites of Dogs. Ackert and Payne carried on their experiments in the Island of Trinidad.

Pathology.—See Parasites of Dogs for general pathology of infestations with this worm. Ackert and Payne report that 3 days after feeding approximately 3000 infective larvae to a pig the animal refused to eat and vomited twice, remaining quiet during that day. The next day and thereafter the pig appeared normal. See *S. suis.*

Treatment and prophylaxis.—See Parasites of Dogs and *S. suis.*

ASCARIS LUMBRICOIDES Linnaeus, 1758

The human and swine ascarid

Synonyms.—*Ascaris suum* Goeze, 1782; *Ascaris suilla* Dujardin, 1845.

Hosts.—Swine, also man and monkey, and, rarely, sheep, dog, and according to Baylis and Daubney, squirrels (*Ratufa indica, Tomeates pygerythrus,* and "squirrel"). Larval stages may develop in guinea-pigs,

Fig. 25. *Ascaris lumbricoides.* *a*, male; *b*, female. Four-ninths natural size. From Neveu-Lemaire, 1912.

rabbits, rats, mice and goats. It has long been debated as to whether the human and the swine ascarid were identical or separate species.

There are no constant dependable differences yet discovered, and present opinion favors the idea that they are identical. Bakker has recently made a careful comparison of ascarids from man and swine and regards them as identical. Barker finds their chromosomes identical.

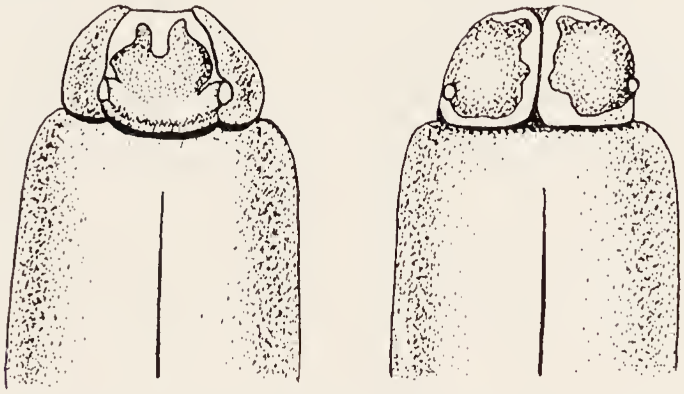


Fig. 26. *Ascaris lumbricoides.* Head end. Left, dorsal view; right, ventral view. x 20. From Neumann, 1883.

Location.—Small intestine, usually; wandering specimens in stomach, cecum, ducts of liver and pancreas, gall bladder, esophagus, pharynx, trachea, bronchi, and nares, and, following perforation of the intestine, the body cavity. In man, Pillay has reported Ascaris from a swelling in the scrotum and Kondo has reported it from the pleural cavity with no evidence of a communication with the liver.

Morphology.—*Ascaris*: Large, thick yellow or pink worms (Fig. 25), somewhat smaller, at least slenderer, in swine than in man, as a rule. Mouth with a dorsal lip and 2 latero-ventral lips (Fig. 26); 2 large, lozenge-shaped papillae, with double terminations, on the dorsal lip near its lateral margins; and 1 large, double papilla towards the ventral side, and 2 small, simple papillae towards the opposite side on each of the latero-ventral lips; margins of lips finely dentate.

Male 15 to 17 cm. long in swine (15 to 25 cm. in man) by 3 to 3.2 mm. wide. Posterior end conical and bent ventrally (Fig. 27). Two spicules, 2 mm. long, not alate, curved ventrally, flattened dorso-ventrally, and widened at their tips. Caudal papillae 70 to 75 in number, of which 7 pairs are postanal and 1 unpaired papilla just before the cloacal aperture. According to Baylis and Daubney, characteristic features are the presence of 2 pairs of large double papillae behind the cloacal aperture and 3 small simple papillae, arranged in a triangle, posteriorly; there is a pair of double preanal papillae in the preanal series.

Female 20 to 25 cm. long in swine, exceptionally up to 40 or 44 cm., (20 to 40 cm. in man) by 5 to 5.5 mm. wide. A swine ascarid in the collection of the Bureau of Animal Industry is 39.8 cm. long by 6 mm.

wide. Guillermet reports one human ascarid 35 cm. long by 1 cm. wide, and one specimen 25 cm. long by 8 mm. wide. Tail straight and conical (Fig. 27). Vulva at juncture of anterior and middle thirds, in

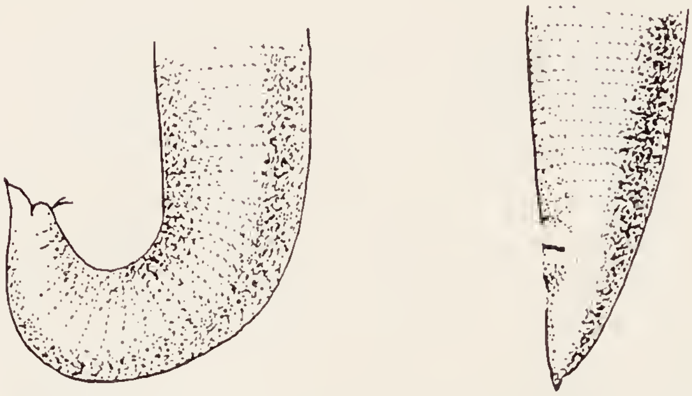


Fig. 27. *Ascaris lumbricoides.* Left, male tail, lateral view, enlarged; right, female tail, lateral view, enlarged. From Neumann, 1883, after Leuckart.

an annular depression. Eggs elliptical, with thick, transparent shells surrounded by a thick layer of albumen which is irregularly mammillated and yellow (Fig. 28); the normal egg is about 64μ long and not yet segmented when deposited. However, egg measurements range from 56.5 to 87.5μ in length and from 46.5 to 57.5μ in width, the length varying much more than the width.

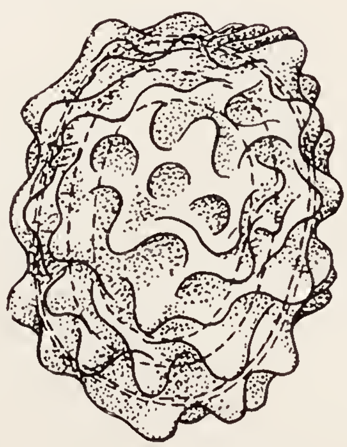


Fig. 28. *Ascaris lumbricoides.* Egg, Enlarged. From Ward, 1907, after Stiles.

Life history.—The eggs pass in the feces and embryos develop in them in the course of 10 to 40 days under favorable conditions of temperature and moisture. When such eggs are swallowed by swine (Fig. 29) or other suitable animals, the embryo escapes from its shell, this occurring in the lower part of the small intestine and in the cecum, according to Fuelleborn, and not in the stomach, in the case of this species. He states that eggs of *Belascaris marginata* do hatch in the stomach. The first stage larva migrates to the liver, lungs, spleen and other organs; the worms which ultimately get to the lungs leave the blood stream and enter the air passages, making their way up the

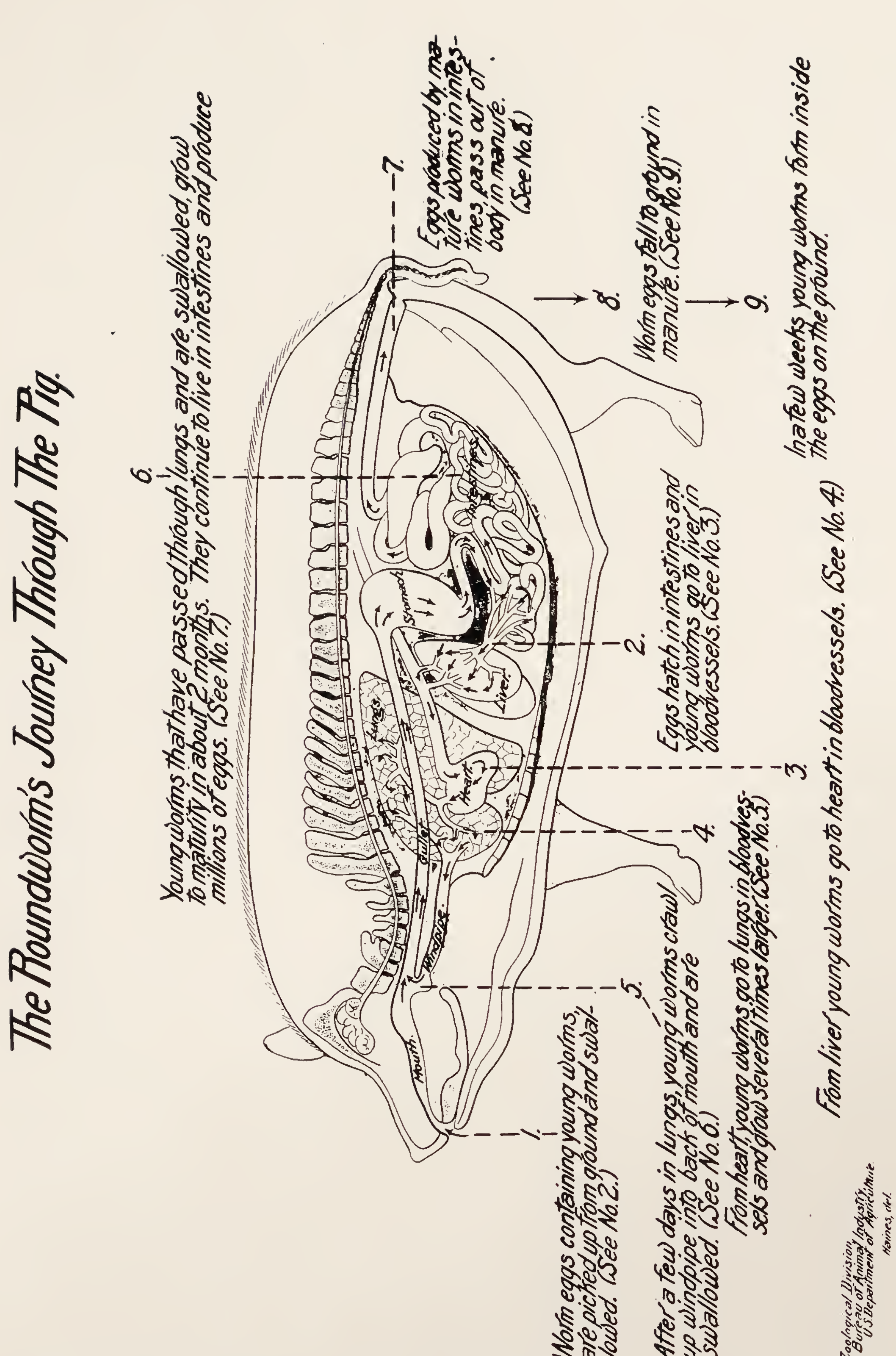


Fig. 29. *Ascaris lumbricoides.* Diagram showing the course of migration of ascarid larvae in swine. From Ransom, 1922.

bronchi and trachea to the pharynx, and pass down the esophagus to the intestine. According to Ransom and Cram, larvae may appear in the portal vein in 17 hours after infection in experiment animals, and may still be passing to the liver 2 days after infection, reaching the portal vein from venules in the intestinal wall or coming by way of the lymphatics and passing the mesenteric lymph nodes. In the liver they pass from the interlobular veins to the intralobular veins. They may reach the hepatic veins in 17 hours or remain in the liver 4 to 6 days. From the right side of the heart they pass to the lungs in 17 hours or may require a much longer time to reach the lungs. They commonly reach the lungs in 7 to 10 days, usually reaching the intestine in 10 days. Yoshida and also Asada believe the larvae usually penetrate the intestinal wall to the body cavity, traverse the diaphragm and enter the liver through the capsule or penetrate the diaphragm and enter the lungs from the pleural surface, but Ransom and Cram could not confirm this. Fuelleborn regards larvae in the body cavities as those which have bored out of capillaries and gone astray. During the course of their migrations, the larvae undergo considerable development and increase to a size of 1.5 mm. or more in length; in the lungs, trachea and pharynx they are 700μ to 1.2 mm. long (Fig. 30). In the intestine they become mature in the course of 10 weeks and mate. Ransom and Foster infected animals by introducing eggs containing embryos under the skin, and Asada reports that he has successfully infected animals by the cutaneous route with larvae hatching in feces.

In Kronecker's solution (physiologic sodium chlorid solution containing .06 gm. of sodium hydroxid per liter), the adult worms have been kept alive up to 26 days, and in physiologic saline up to 15 days, at 25.5 to 30 degrees C. by Hall.

Kondo is reported in an abstract to have found that ascarid eggs hatch in soil and feces in the course of 70 days and that the larvae live over 1 month in soil and over 2 months in feces. It is further stated that when smeared on the skin of young guinea pigs, these larvae penetrated the skin and were found in the lungs and liver. These larvae lived 2 to 9 hours in gastric juice and developed when fed as free larvae as well as when fed in eggs.

Distribution.—Cosmopolitan; abundant in swine in the United States.

Pathology.—The presence of these worms in the intestine in large numbers has been credited with the production of more or less enteritis, usually of a low grade, catarrhal nature, accompanied by digestive trouble and malnutrition, and a limited amount of critical work indi-

cates quite definitely that heavily infested animals, especially young pigs, do not make the growth or attain the condition of animals under similar conditions, but uninfested. Swine infested with worms may be in good condition, but would probably be in better condition without them. In one experiment conducted by Dr. H. B. Raffensperger, infested swine 6 months old weighed about half as much as uninfested swine of the same age and raised under the same conditions. Experiments indicate that young animals are particularly susceptible to infestation, in addition to suffering more from the effects of the worms. Garin states that ascarids feed on epithelial cells in the intestine, and the mouth structure is adapted to such feeding habits. The worms have been accused of being blood-suckers, but this is debatable; I have found blood in ascarids in the dog associated with marked hemorrhagic gastro-enteritis. In addition, the worms determine other more or less serious conditions. In very large numbers they may cause occlusion

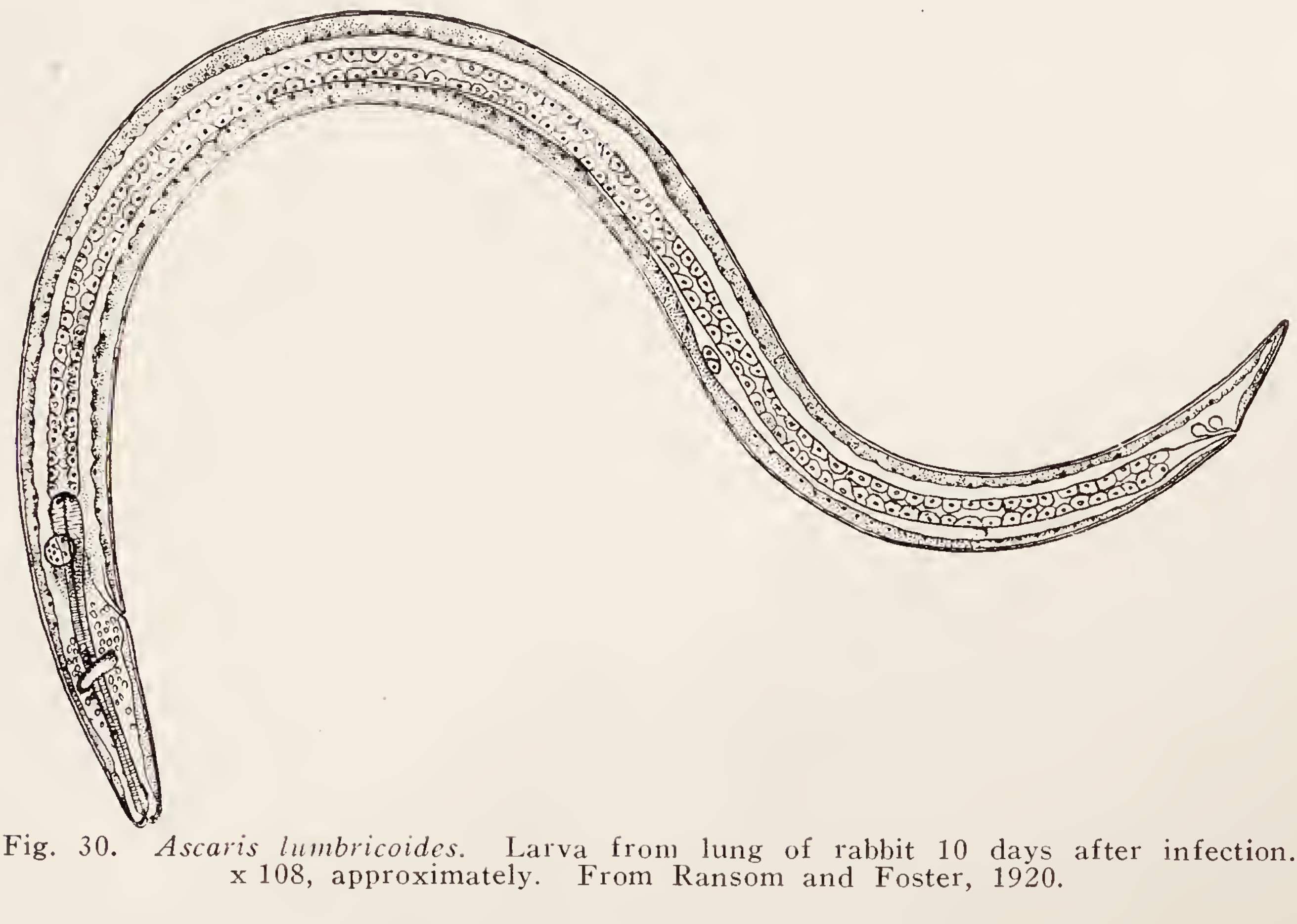


Fig. 30. *Ascaris lumbricoides.* Larva from lung of rabbit 10 days after infection. x 108, approximately. From Ransom and Foster, 1920.

or obstruction of the intestine. In such cases there may be anemia, loss of appetite, colic, epileptoid convulsions and paresis, terminating in the death of the animal. Ascarids are notorious for their wandering habits and may get into the ducts of the liver and pancreas and into the gall bladder with serious results at times. They are usually alive in these sites but are occasionally dead and macerated. Infested livers

may have the characteristic ascarid odor. Occlusion of the ducts may cause icterus. The worms in the ducts cause catarrhal and purulent inflammation of the mucosa of the ducts, according to Joest, not infrequently accompanied by ulceration. Joest attributes to chemical destruction from absorbed ascarid products, rather than to mechanical action, the loss of the superficial epithelium, the partial destruction of the glands and the purulent infiltration of the mucosa. The worms may wander up the esophagus and down the trachea and lodge in a bronchus, causing a gangrenous pneumonia. Poenaru has reported an ascarid from the nasal fossa of a pig showing pronounced nervous and digestive disturbances. In the stomach they may cause a gastritis and in any case are likely to interfere with gastric digestion. In the human female this worm has been found in the Fallopian tube by Nacken. The excretions and secretions of the worms may be toxic, especially under certain conditions. In man, ascarids are believed by Guerrini and by Vestea to play a predisposing role in cholera. The possibility that they may predispose swine to such diseases as hog cholera is worth keeping in mind; the sanitary control measures for worms in swine are of value in the control of hog cholera.

In their customary site in the small intestine, the symptoms of ascarid infestation are those of intestinal parasitism in general—an afebrile condition with digestive disturbances, including diarrhea or constipation, and malnutrition. Diagnosis may be readily made by fecal examination and the finding of the characteristic eggs in the feces. In actual practice it is more likely to be made on the passage of worms or on postmortem examination of animals that succumb to infestation or from other causes.

Recent work has shown that an important phase of the damage attributable to ascarids is due to their activities as larvae in the invasion stage. They cause lesions in the liver and the common parasitic cirrhosis in swine livers may yet be shown to be due, at least in part, to these larvae. In their passage from the blood to the air passages in the lungs, they cause pronounced pathological conditions resulting in localized or general pneumonia. In young pigs, the condition known as thumps is commonly due to larval ascarids in the lungs. On postmortem examination these conditions are usually manifested by the presence of bright-red petechiae or, occasionally, ecchymoses. The injured regions may become infected with pyogenic bacteria. These conditions have long been confused with the lesions of hog cholera and swine plague, and in the absence of characteristic febrile conditions have been a source of perplexity and uncertainty. In heavy in-

festations, these invasions by the larvae may cause death. It is therefore important to keep this possibility in mind in dealing with obscure pneumonias in swine. At the right period, about a week after swallowing the infective eggs, the larvae may be found on microscopic examination of the areas showing the petechiae. Heavy infestations may cause marked pathological alterations in the liver. Hoeppli reports finding a larva in one of the lateral ventricles of the brain in an experiment animal, and Fuelleborn reports lesions of the kidneys and other organs in the dog from larvae of dog ascarids.

Usami and Kamada report obtaining complement fixation in using the body fluid of ascarids and the serum from cases of ascariasis, as well as from cases of ancylostomiasis. Hence the complement fixation is not specific. Their best antigen was a saline extract of female worms. Schwartz finds that these worms contain a hemolysin which is thermostabile, non-specific and alcohol-soluble. It appears to be rather firmly bound to the cells of the worms, presumably to the intestinal cells where it is elaborated.

Hemolysis is not due to fatty acids alone, since the fraction of extract left after ether extration is hemolytic. The hemolysin is neutralized by normal blood serum. The body fluid of the worms when fresh from the host contains oxyhemoglobin and is non-hemolytic. After the worms have lived *in vitro* a few days the body fluid loses its oxyhemoglobin and becomes hemolytic. These worms contain a hemagglutinin which is soluble in salt solution and active at temperatures of 6 to 8 degrees C. The worms also contain a substance, of comparatively slight potency, which inhibits the coagulation of blood.

In view of the fact that the veterinarian's patients cannot describe to him in detail many of the symptoms accompanying heavy infestations with ascarid larvae, it seems worth while to quote extensively from a paper by Koino, describing the symptoms in artificial infestations in man, in order that we may have this experience as a clue to what occurs in animals. Koino's brother, a man 21 years old, swallowed 500 eggs of swine ascarids, and Koino himself, an older man, swallowed 2000 eggs of human ascarids.

In the case of the person taking 500 swine ascarid eggs, the patient's temperature on the third day was 37.4 degrees C; up to the eighth day, the morning temperature was 36.4 to 36.9 degrees, and the evening temperature somewhat higher, 37 to 37.5 degrees. The ninth day the temperature rose to 39.1 degrees following a severe chill. There were sharp headache, cough, pain in chest, anorexia, general fatigue and difficult breathing. The next 3 days the temperature was 37.3 to 39

degrees, the next day it was 37 degrees, and on the seventh day after the onset of the fever it returned to normal. The cough gradually became worse, with an increased amount of watery sputum containing no blood. There were pains and a sense of heaviness in the chest. Respiration increased to 27 to 32 per minute, returning to normal 5 days after the onset of the fever. Chest rales were audible from the second to the sixth day. Symptoms disappeared on the eighth day. No ascarid larvae were found in the sputum. No worms were recovered by anthelmintics 50 days after taking the eggs.

In the case of the person taking the 2000 human ascarid eggs, the temperature rose to 37.2 degrees on the third day and gradually became higher. On the sixth day there was fever, following chills, with severe headache, increased respiration and pulse, flushed face, thirst, heavy feeling over chest, and cough with large amounts of sputum. The temperature varied from 38.7 to 42 degrees, falling to normal on the ninth day. On the fifth and sixth day, there was severe respiratory difficulty, rate 56 to 58 per minute, and the face was cyanotic. From the seventh day the number decreased, being 24 on the ninth day and about normal on the sixteenth. The pulse went to 90 to 120 per minute. The cough subsided on the eleventh day. The amount of sputum increased to 155 cc. on the fifth day and was not collected on the sixth and seventh day owing to the serious condition of the patient. Blood was present on the fifth and sixth day. There was no appetite while the patient's condition was serious; appetite was about normal on the ninth day after the onset of the fever. There was severe lumbago on the third and fourth days and pains in the gastrocnemius muscles on the fourth and fifth days. There was a marked dislike for the odors of bedding, soap, towels and perfumes. Rales were audible on the fourth day and persisted through the ninth. The liver was enlarged and palpable on the fourth day, slightly palpable through the twelfth, and not palpable on the fifteenth. One larva was collected from the sputum on the third day, 5 the fourth, and 178 the fifth. No collections were made the next 2 days owing to the patient's condition. The eighth, ninth and tenth days, there were 16, 7 and 5 larvae present; none found thereafter. A total of 667 worms were recovered by anthelmintics 50 days after taking the eggs.

These cases show the production of enlarged liver, pneumonia, headache, fever, dyspnoea, cough, muscular pains, and hemoptysis in the course of a case of infestation with larval ascarids.

Magary Kossa thinks ascarids in swine give rise to gastro-intestinal atony, and this to gastric stasis and hypertrophy, occlusion and inanition, with catarrhal conditions and retention of feces.

Treatment.—The treatment which has been found most satisfactory in actual tests of various anthelmintics, where treatment has been followed by careful examination of the feces after treatment and finally by post-mortem examination of the experiment animals to ascertain the number of worms not removed, consists in the administration of oil of American wormseed, at a dose rate of 1 fluid dram to a 100-pound animal preceded or followed immediately by 2 fluid ounces of castor oil or given in the oil. Doses for animals of various sizes may be determined from this basis, though it is likely that a dose of 2 fluid drams is adequate for animals of 300 or 400 pounds weight. The drugs may be given with a dose syringe or by stomach tube. The swine should be fasted for 18 to 24 hours and not fed or watered for 3 hours after treatment. Some veterinarians substitute calomel or aloes for castor oil; others use Glauber's salt or Epsom salt, at the rate of 1 pound to 10 hogs, in solution on oats or in slops 3 hours after treatment. Carbon tetrachlorid at a dose rate of 1 fluid ounce in 3 fluid ounces of castor oil is slightly less effective than chenopodium and is rather bulky; it does not appear to be well adapted to use in the case of swine, since it seems to affect the liver to some extent and swine commonly suffer from parasitic cirrhosis of the liver, a condition which leaves relatively little functional tissue. Santonin is used in doses up to 8 grains, but is expensive and in single dose is less effective than chenopodium.

There is a persistent demand for a treatment that can be administered in slops to a number of swine, but while this would doubtless be much easier, experiments up to the present time indicate that it is not successful and that individual treatment under favorable conditions is essential for satisfactory results. Similarly, the numerous mineral mixtures that have been advocated as removing worms or preventing worm infestation are not found effective for this purpose on critical test. At the same time, these mixtures may be of some value in furnishing mineral constituents of value in the economy of the swine, and may have a tonic value. They should be fed for this purpose, if used, and not used as anthelmintics.

Numerous capsules have been marketed as anthelmintics for removing ascarids from swine. In a large number of cases their use has been followed by the deaths of the animals treated. This is due to the fact that the capsule is lodged in the pharyngeal pouch (retropharyngeal recess), causing severe inflammation, swelling of the neck and

suffocation. If capsules are placed on the back of the tongue, they are often rejected. Some veterinarians use a speculum and pass a stomach tube in giving drenches to swine. With a tube the size of a horse catheter it is not very difficult to dose swine in this manner.

Prophylaxis.—This is a matter of sanitation and cleanliness. As has been said, swine should be kept in a less swine-like manner than is customary. The maintenance of filthy, muddy sties and wallows favors the development of these worms and leads to losses. Swine should receive the same care as other animals in the provision of clean quarters that can be kept clean. Manure must be frequently and thoroughly removed and properly disposed of. Since it appears that the swine ascarid is identical with the human ascarid, contamination of the food, water and surroundings of swine by human feces must be prevented, and, similarly, the contamination of man's food, water, hands, etc., by swine manure must be regarded as dangerous in the conveyance of this worm as well as otherwise aesthetically objectionable and dangerous. Since young pigs are the chief sufferers and may become infested by suckling, from eggs in dirt on the sows' teats, sows should be kept in clean, dry places away from mud wallows, before farrowing, and the sows and pigs kept in clean quarters until the pigs are 4 months old at least. We owe most of our knowledge of ascariasis in swine, as regards the life history of the worm responsible for it, the importance of the disease, and the methods of prevention, to Ransom.

Under the McLean County System of swine sanitation as developed in Illinois under the supervision of the Federal Bureau of Animal Industry, sows are thoroughly washed and scrubbed just before farrowing and placed in farrowing pens which have been thoroughly cleaned with hot water, soap and lye. Within 10 days after farrowing, the sows and litters are hauled in crates, by sled or wagon, to a clean field and kept there until the young pigs are at least 4 months old. By this time they have acquired resistance to infection with ascarids and to the effects of infestation. By this system the losses among young pigs from pulmonary and intestinal ascariasis, as well as from such conditions as "bull nose," are avoided. This saving and the increased growth amply pays for the involved expense and trouble.

As is usually true regarding innovation and improvement, especially where it involves expense, many farmers are opposed to cleanliness in the surroundings of swine, and regard it as unnatural and even objectionable to the swine. This attitude carries its own penalties in the form of damage done by worms and disease of various sorts. The

demonstration of the financial advantage of cleanliness may be depended on to lead to real improvement.

The success of this system has directed attention to the fact that parasitism is most important as a form of disease in young animals and that the high infant mortality among our livestock is largely due to parasitism. It has demonstrated that much of our losses among young livestock may be prevented by giving special care to these young animals, and that sanitation is of major importance as a substitute in youth for the acquired immunity to many parasites that comes with age to older animals. It shows that the specific measures that serve to prevent parasitic infestation also serve in a large measure to prevent many other diseases. This work in swine sanitation may prove to be the starting point for a general improvement in the handling of young livestock, a replacement of our present attitude of indifference towards the deaths of young animals, an attitude the writer has denominated the doctrine of infant damnation of livestock, by an attitude more comparable to our present attitude towards human infant mortality as a thing largely preventable by proper care, the doctrine of salvation by sanitation.

ŒSOPHAGOSTOMUM DENTATUM (Rudolphi, 1803) Molin, 1861

The nodular worm of swine

Synonym.—*Strongylus dentatus* Rudolphi, 1803.

Hosts.—Swine and wild boar. Grosso has reported this species from an ape, *Macacus sp.*, but it seems probable from his figure that he had another species.

Location.—Large intestine, as adults; in walls of intestine, not forming conspicuous nodules, as larvae. First reported from liver by Leidy and not uncommonly found there in a degenerated condition in cysts in meat inspection.

Morphology.—*Œsophagostomum*: White or grey-brown opaque worms. Buccal capsule broader than long (Fig. 31), the mouth opening directly forward and bordered by a crown of numerous, small, pointed processes; outside of this, 6 papillae. Cervical papillae opposite the posterior fourth of the esophagus. No lateral alae. Anterior extremity with a short cuticular peristomal collar separated by a circular cephalic groove from a larger cuticular swelling, the cephalic vesicle, which is terminated abruptly by a transverse cleft. Buccal margin with external leaf crown; buccal capsule with short internal leaf crown.

Male 8 mm. to 1.2 cm. long. Spicules slender, 1.13 mm. long. Caudal bursa vaguely trilobed (Fig. 32). Dorsal rays bifurcated. Postero-lateral and medio-lateral rays close together and parallel. Ventral rays close together and parallel.

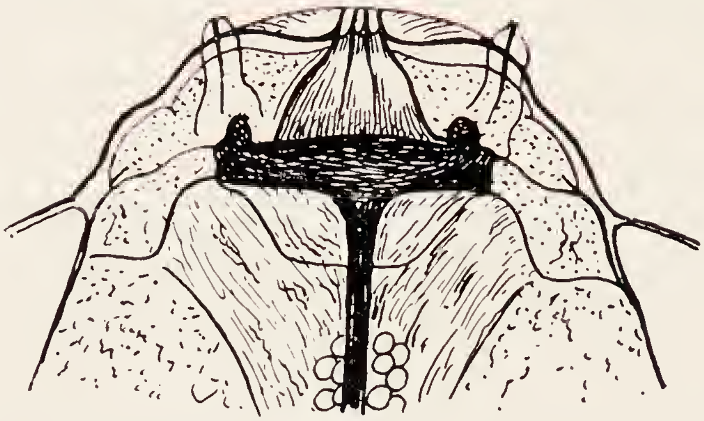


Fig. 31. *OEsophagostomum dentatum.* Head. Enlarged. From Fiebiger, 1912.

Female 1.2 to 1.5 cm. long. Tail subulate and mucronate. Vulva just anterior of the anus and surrounded by chitinous lips. Eggs 60 to 80μ long by 35 to 45μ wide (Fig. 33).

Fig. 32. *OEsophagostomum dentatum.* Bursa. x 90. From Schneider, 1866.

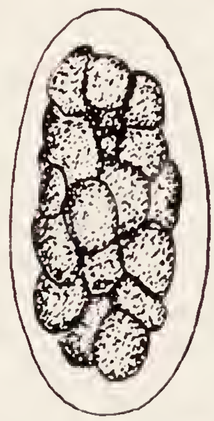


Fig. 33. *OEsophagostomum dentatum.* Egg. Enlarged. From Dimock, 1921.

Embryo 254 to 272μ long.

Life history.—Eggs produced by the female worms pass in the manure and hatch, the larvae developing to the infective stage. When such infective larvae are swallowed by swine the larvae penetrate the intestinal wall and form small intestinal nodules. According to Olt, the larvae in the nodules are 1.7 to 5 mm. long. We have found them 6 mm. long or longer. In view of the fact that these larvae attain a length 50 per cent greater than do the larvae of *Proteracrum columbianum* (3 to 4 mm.) and still produce only small nodules as compared

with those of *P. columbianum* in sheep, it would appear that the bacterial invaders are much less effective and the intestine more resistant to them in the case of swine than in the case of sheep. The larvae escape from the nodules to the lumen of the intestine and become mature worms. Some larvae go astray and lodge in the liver where they degenerate.

Distribution.—United States, Porto Rico, British West Indies (Antigua), South America, Europe, Asia and Zanzibar.

Pathology.—The adults in the lumen of the large intestine are not known to do any noticeable damage, and the nodule-forming stage, similar to that of *Proteracrum radiatum*, causes comparatively small nodules, usually of pin-point to pin-head size. According to Dimock, these worms may cause unthriftiness, intestinal irritation, weakness, anemia, constipation followed by diarrhea, and possibly a few deaths. Temperature normal or increased. Intestine somewhat congested or inflamed. The presence of these worms in the liver, associated with necrotic material in cysts, causes some loss of livers in packing houses.

Treatment.—Unknown. Hall and Shillinger did not find carbon tetrachlorid valuable in removing these worms.

Prophylaxis.—Sanitary measures, suitable manure disposal, and cleanliness are the indicated measures for the control of this worm. See *Ascaris lumbricoides,* page 60.

BOURGELATIA DIDUCTA Railliet, Henry and Bauche, 1919

Bourgelat's nodular worm

Hosts.—Swine.

Location.—Cecum and colon.

Morphology.—*Bourgelatia*: Body whitish, often sinuous, about 20 to 24 times as long as wide, attenuating towards the extremities and of almost uniform width throughout the middle three-fifths of body. Cuticula transversely striated, the intervals being 5 to 7μ near the middle of the body. Anterior extremity truncated and rounded. No cephalic vesicle and no transverse ventral cleft in cervical region. Buccal capsule wider than long, with thick wall, joined to the esophageal armature; the point of union is marked by a crenulate or denticulate line representing a crown of small quadrangular scales (Fig. 34). External leaf crown formed of about 20 large, pointed, convergent elements. The internal leaf crown has about twice as many elements, less pointed and doubled on their external surface by very refringent, twinned projections. Submedian papillae extend slightly beyond the cuticula of

the posterior peristomic segment. Cervical papillae feebly developed, with a hemispherical base almost entirely concealed in the cuticula, only the fine central point extending out. Esophagus slightly swollen at its origin, thinning in the second fourth of its length, then widely dilated in pestle shape in the posterior half; the 3 faces of its lumen have a strong corneous lining. At its origin the intestine is smaller than the esophagus and its cells are often pigmented black, especially in the anterior portion.

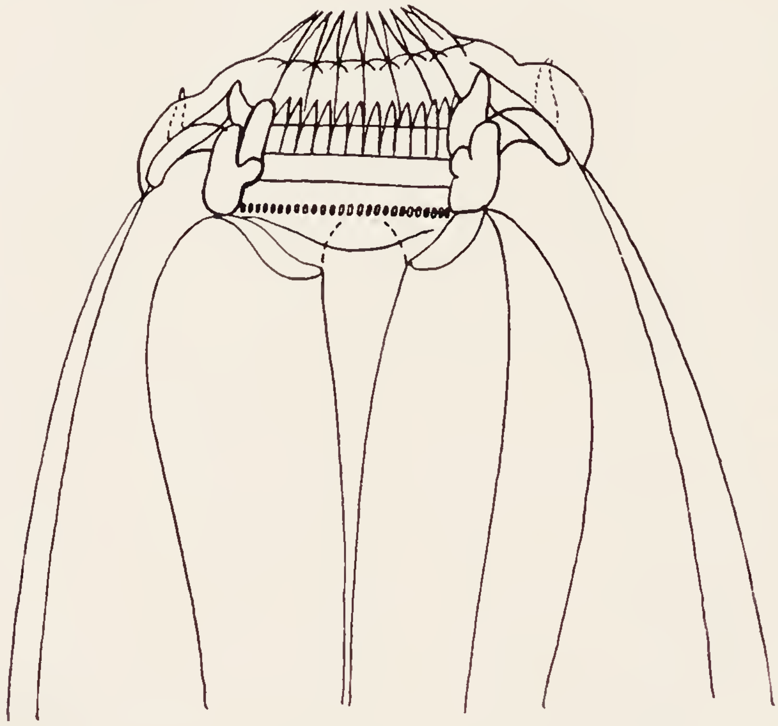


Fig. 34. *Bourgelatia diducta*. Anterior end. Dorsal view. x 200. From Railliet, Henry and Bauche, 1919.

Male 9.3 to 12.5 mm. long by 400 to 600μ wide. Esophagus 650 to 760μ long by 135 to 170μ wide anteriorly and 260 to 360μ posteriorly. Cervical papillae 420μ from the anterior end. Caudal bursa (Fig. 35) with a small dorsal lobe, 2 much larger lateral lobes, and 2 quite prominent ventral lobes. The dorsal, postero-lateral, mediolateral and ventral rays extend to the border of the bursa. The external branch of the dorsal ray branches from the dorsal ray in a relatively open angle. The lateral rays are divergent. Spicules winged, the wing finely striate transversely, uniting to pass through a gubernaculum, and curving dorsally towards the distal extremity; spicules 1.25 to 1.33 mm. long; gubernaculum 135μ long.

Female 11 to 13.5 mm. long by 500 to 640μ wide. Esophagus 750 to 850μ long by 165 to 200μ wide anteriorly and 260 to 300μ wide posteriorly. Cervical papillae 540 to 550μ from anterior end. Posterior extremity (Fig. 36) attenuated, beginning anterior to the vulva region;

tail pointed. Anus 400 to 425μ from the tip of the tail and on a slight elevation. Vulva 950μ to 1.06 mm. from the tip of the tail and on a prominent cylindrical elevation. Total length of ovejectors 200 to 230μ. Caudal papillae asymmetrical, 110 to 140μ from the tip of tail. Eggs ellipsoidal, 69 to 77μ long by 38 to 42μ wide, in the morula stage when deposited.

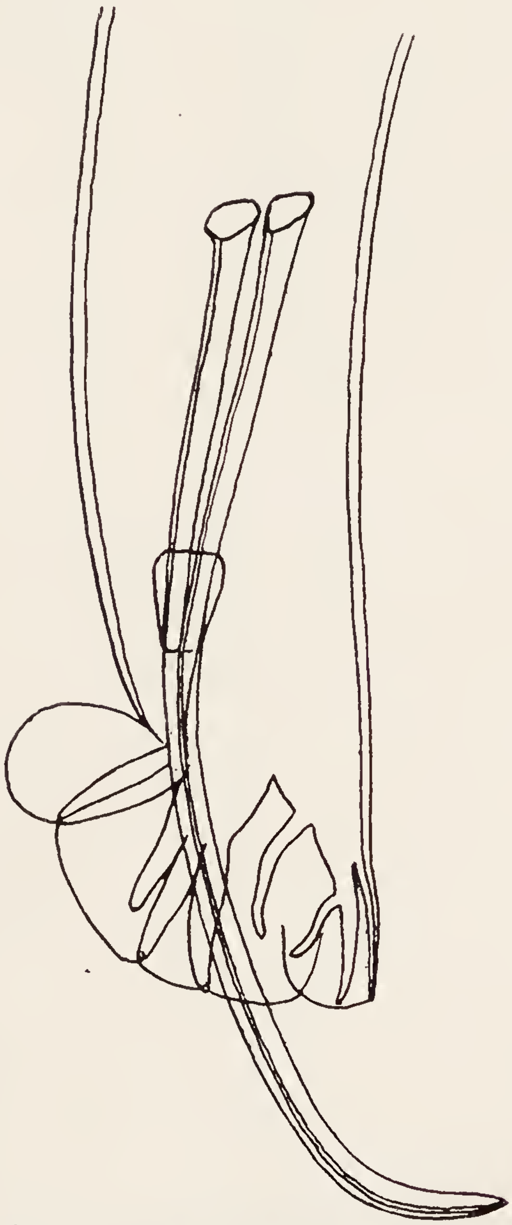


Fig. 35. *Bourgelatia diducta.* Male tail. Lateral view. x 75. From Railliet, Henry and Bauche, 1919.

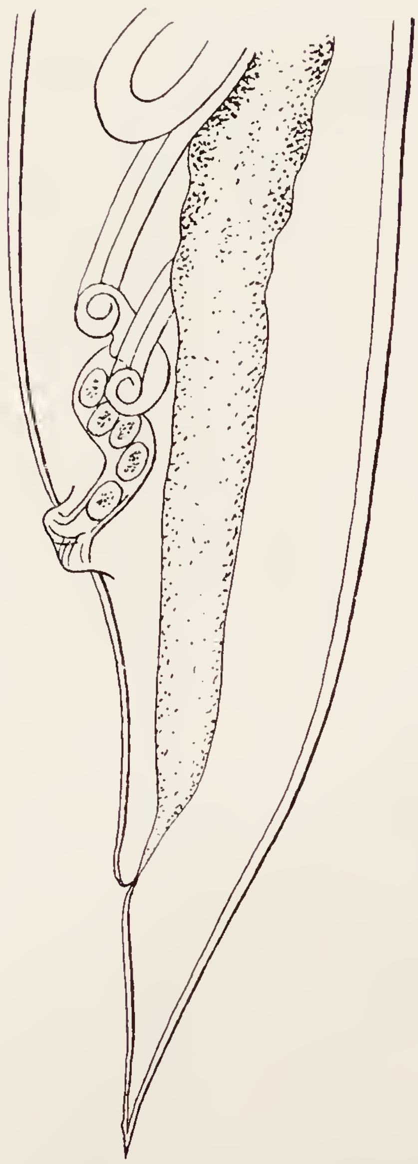


Fig. 36. *Bourgelatia diducta.* Female tail. Lateral view. x 50. From Railliet, Henry and Bauche, 1919.

Life history.—Unknown; judging from the morphological relationships of this worm, the life history would be similar to those of nodular worms in general.

Distribution.—Annam (Hué).

Pathology.—Unknown; we may surmise that it is similar in a general way to that of some of the nodular worms.

Treatment.—Unknown; what has been said elsewhere with reference to treatments for nodular worms in swine may apply here.

Prophylaxis.—Unknown; the prophylactic measures recommended in the case of the common nodular worm of swine would probably be of value here.

Railliet, Henry and Bauche note that Rudolphi may have had specimens of this worm, judging from his description of *Strongylus dentatus,* now *Œsophagostomum dentatum.*

GLOBOCEPHALUS LONGEMUCRONATUS Molin, 1861

The globe-headed worm

Synonyms.—*Cystocephalus longemucronatus* (Molin, 1861) Railliet, 1895; *Characostomum longemucronatum* (Molin, 1861) Railliet, 1902.

Hosts.—Swine.

Location.—Small intestine.

Morphology.—*Globocephalus*: Buccal capsule (Fig. 37) spherical, wider than the border, with 2 chitinous rings, 1 at the base and 1 at the aperture connected by 4 ribs; its anterior aperture has a simple circular margin without teeth and is only bent slightly toward the dorsal face; no ventral teeth. Esophagus long and almost cylindrical, slightly enlarged distally and surrounded at this point by a circlet of hemispherical glands.

Male 7 mm. long by 200μ wide. Bursa with 3 lobes (Fig. 37). Dorsal ray with 2 branches, each terminating in 3 digitations. Externodorsal rays arise near the base of the dorsal trunk. Lateral and ventral rays arise from a common trunk, the ventral set separating from it has almost parallel rays, the lateral branching almost at the same level with one another and slightly divergent from one another. Molin figures a knob on the dorso-lateral ray near its base. The cloacal aperture opens on the truncated end of the genital cone. There are 2 slender transversely striate spicules with a double curve near the tips, and a gubernaculum described as trowel-shaped, but which, from Molin's figure, is very similar to that of *Crassisoma urosubulatum.*

Female 8 mm. long by 300μ wide. Tail ends in a short conical point (Fig. 38). Vulva prominent, in the posterior half of the body at some distance from the anus. (As figured by Molin, the vulva is scarcely prominent.) The anus is at some distance from the tip of the tail and partly covered by an anterior lip. Vagina piriform, perpendicular to body wall. Ovejectors divergent.

Life history.—Unknown. See *Crassisoma urosubulatum*, page 73.

Distribution.—Austria and Gold Coast (fide Macfie).

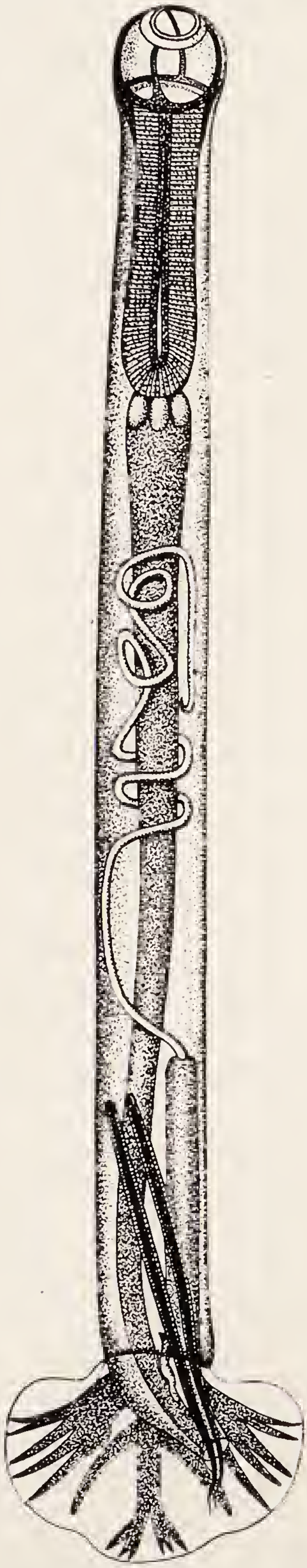


Fig. 37. *Globocephalus longemucronatus.* Male worm. Ventral view. Enlarged. From Molin, 1861.

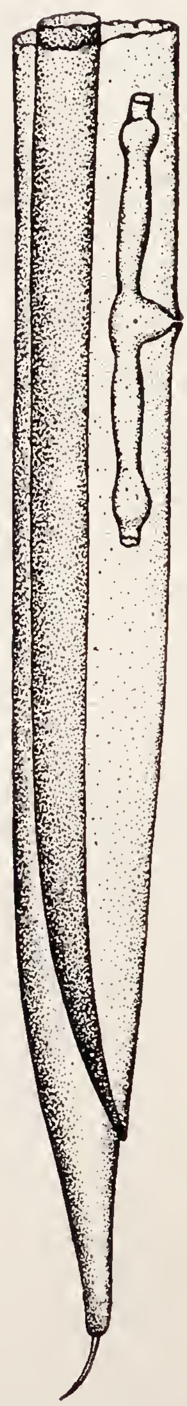


Fig. 38. *Globocephalus longemucronatus.* Female tail. Lateral view. Enlarged. From Molin, 1861.

Pathology.—See *C. urosubulatum,* page 74.

Treatment.—Unknown; see *C. urosubulatum,* page 74.

Prophylaxis.—Probably capable of control by simple measures of sanitation, proper manure disposal, and general cleanliness about yards and buildings occupied by swine. See *C. urosubulatum,* page 74.

There is a possibility that this worm is identical with *Crassisoma urosubulatum* (See page 74).

GLOBOCEPHALUS CONNORFILII Lane, 1922

Connor's globe-headed worm

Hosts.—Swine.

Location.—Not given; probably intestine.

Morphology.—*Globocephalus*: Stout, short worms, usually curved with the concavity dorsal. Oral aperture unarmed, dorso-subterminal; oral capsule (Fig. 39) not globular, but elongate antero-posteriorly, and provided with a pair of rudimentary basal subventral teeth projecting into the oral cavity; capsule has 2 transverse thickenings (Fig. 40), of which one is close to and parallel to the almost circular oral aperture, and the other close to the union of the capsule with the esophagus and angular in lateral view. Dorsal esophageal gland opens inconspicuously into oral capsule in mid-dorsal line. Nerve ring and cervical papillae near middle of esophagus.

Fig. 39. *Globocephalus connorfilii.* Head. Lateral view. From Lane, 1923.

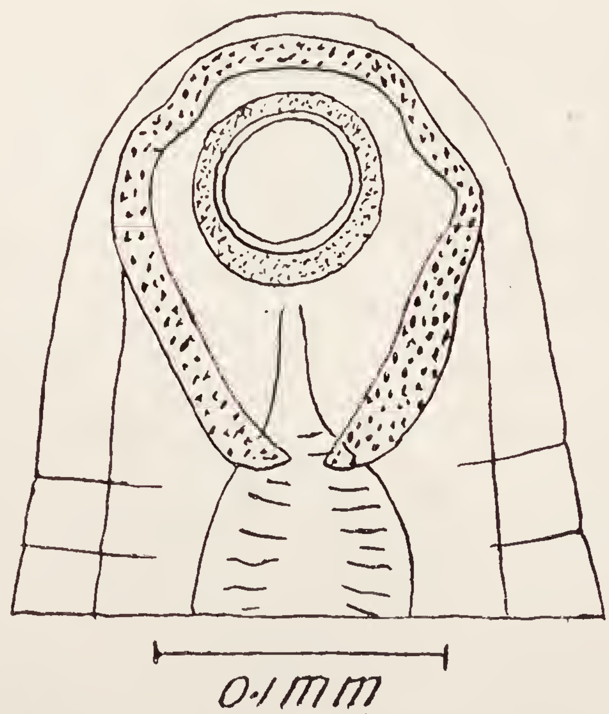


Fig. 40. *Globocephalus connorfilii.* Head. Dorsal view. From Lane, 1923.

Male 4.5 mm. long by 275μ wide. Dorsal lobe of bursa small (Fig. 41). Dorsal rays united for almost three-fourths of their length; after separation each ray branches almost immediately into an external thin branch and an internal thick branch, the latter again dividing. The

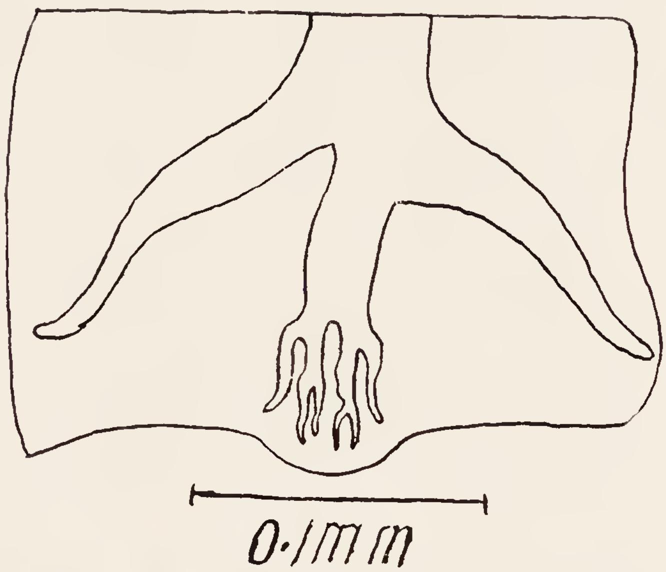


Fig. 41. *Globocephalus connorfilii.* Dorsal and externo-dorsal rays of bursa. From Lane, 1923.

Fig. 42. *Globocephalus connorfilii.* Bursa. From Lane, 1923.

externo-dorsal rays originate asymmetrically from the second fourth of the dorsal ray. The lateral rays (Fig. 42) have a common origin; the dorso-lateral is the stoutest and is separated from the medio-lateral by a deep cleft, both rays having a dorsal trend; the externo-lateral is separated from the medio-lateral by a somewhat shallow cleft and has a slight ventral trend; the ventral rays are apposed. Prebursal papillae long. Spicules (Fig. 43) similar and equal, 275μ long, gently tapering and with their fine rounded tips curved posteriorly. Gubernaculum 90μ long, canoe-shaped in lateral view, kite-shaped in dorso-ventral view.

Female 6 mm. long by 325μ wide. Distance from anterior end to cervical papillae, nerve ring and excretory pore is 375μ. Posterior end of esophagus 750μ from head end of worm. Vulva 2.4 mm. from tip of the tail, the tail (Fig. 44) long and fine-pointed and bearing small caudal papillae. Eggs in uterus 60μ long by 40μ wide.

Fig. 43. *Globocephalus connorfilii.* Gubernaculum and tips of spicules. From Lane, 1923.

Fig. 44. *Globocephalus connorfilii.* Female tail. From Lane, 1923.

Life history.—Unknown; probably similar to that of *Crassisoma urosubulatum* (See page 73).

Distribution.—Samoa (Lane's first paper gives "Pacific" for this).

Pathology, treatment, etc.—Unknown. Probably similar to *Crassisoma urosubulatum* (See page 74).

CRASSISOMA UROSUBULATUM Alessandrini, 1909

The swine hookworm

Synonyms.—*Ankylostomum longemucronatum* (Molin, 1861) Linstow, 1897; *Characostomum longemucronatum* (Molin, 1861) Railliet, 1892; *Cys-*

tocephalus longemucronatus (Molin, 1861) Railliet, 1895; *Crassisoma subulatum* Fiebiger, 1923.

Hosts.—Swine.

Location.—Small intestine.

Morphology.—*Crassisoma:* Body more or less curved. Cuticula transversely striated. Buccal capsule elongate oval (Fig. 45), opening somewhat dorsal; capsule with 8 meridial ribbings, according to Alessandrini, or 14, according to von Linstow, without teeth or chitinous cutting plates at its anterior border, according to Alessandrini (*C. samoense* has cutting plates), but with 2 ventral teeth at base of capsule; dorsal gutter present. Esophagus wider posterior to nerve ring

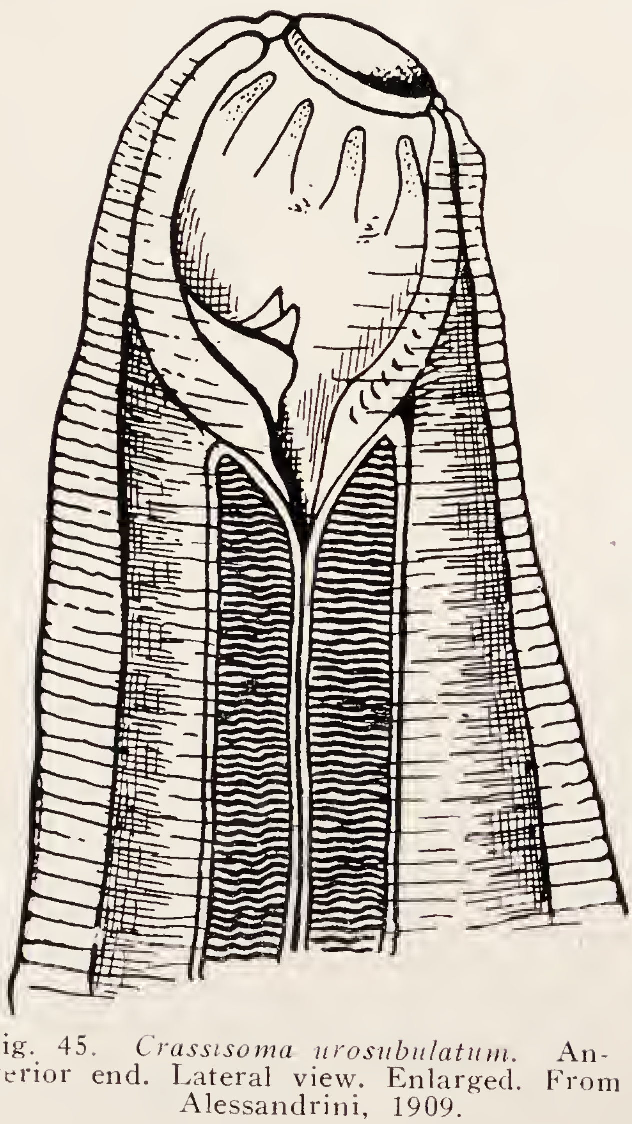


Fig. 45. *Crassisoma urosubulatum.* Anterior end. Lateral view. Enlarged. From Alessandrini, 1909.

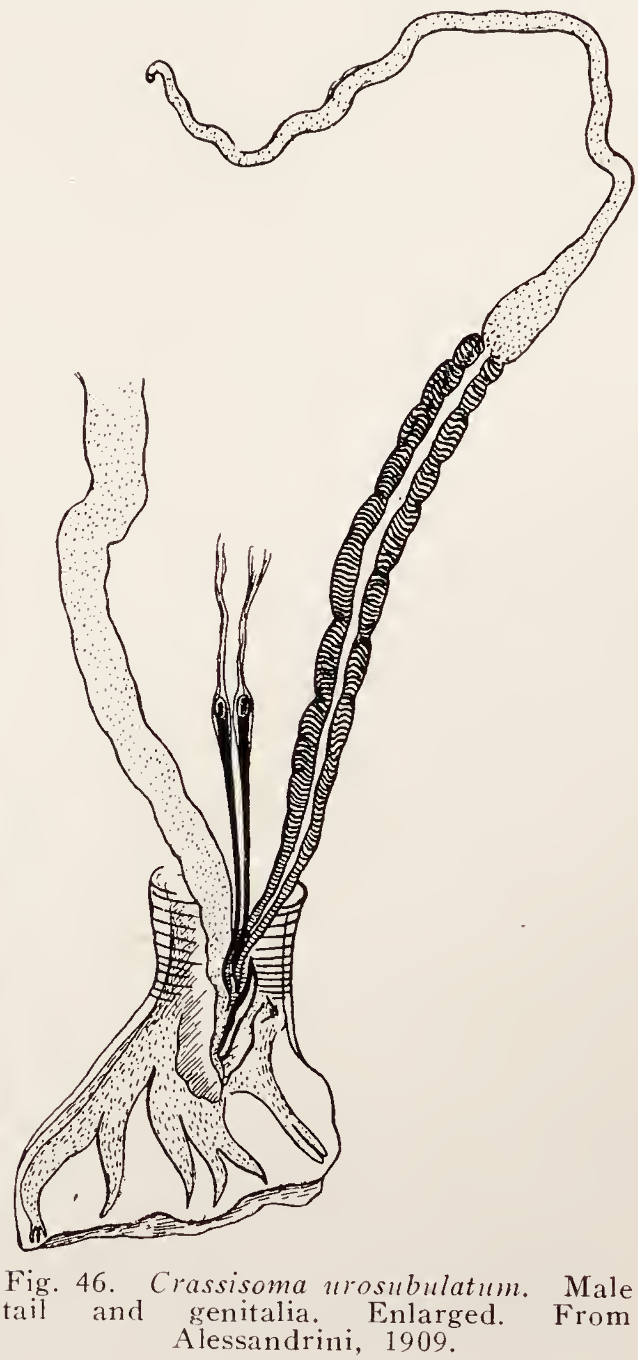


Fig. 46. *Crassisoma urosubulatum.* Male tail and genitalia. Enlarged. From Alessandrini, 1909.

than anterior to it. Two large cervical papillae slightly posterior to nerve ring.

Male 4.4 to 4.58 mm. long by 360 to 380μ wide. Esophagus about 612μ long, or 10/61 of total body length. Bursa (Fig. 46) with large integral latero-dorsal lobe and with a small, semilunar ventral lobe. Ventral rays close together and parallel. Lateral rays originate in a thick, curved common stem and are divergent and slightly wavy; the postero-lateral ray forms the first branch from the common stem. Dorsal ray originates as a very thick stem, which gives off the wide externo-dorsal rays about half-way from the base to the tip, then thins and branches two-thirds of the distance from this point to the tip, the 2 branches terminating in trifid tips. Spicules 580 to 590μ long, slender, irregularly wavy, especially near the tips, transversely striated, each thinning posteriorly to a terminal point. The gubernaculum forms a gutter or channel with irregularly undulate margins and is deeper and wider posteriorly than anteriorly. In the region of the genital cone (Fig. 47), there are 3 mammillate dorsal papillae, with a long clavate

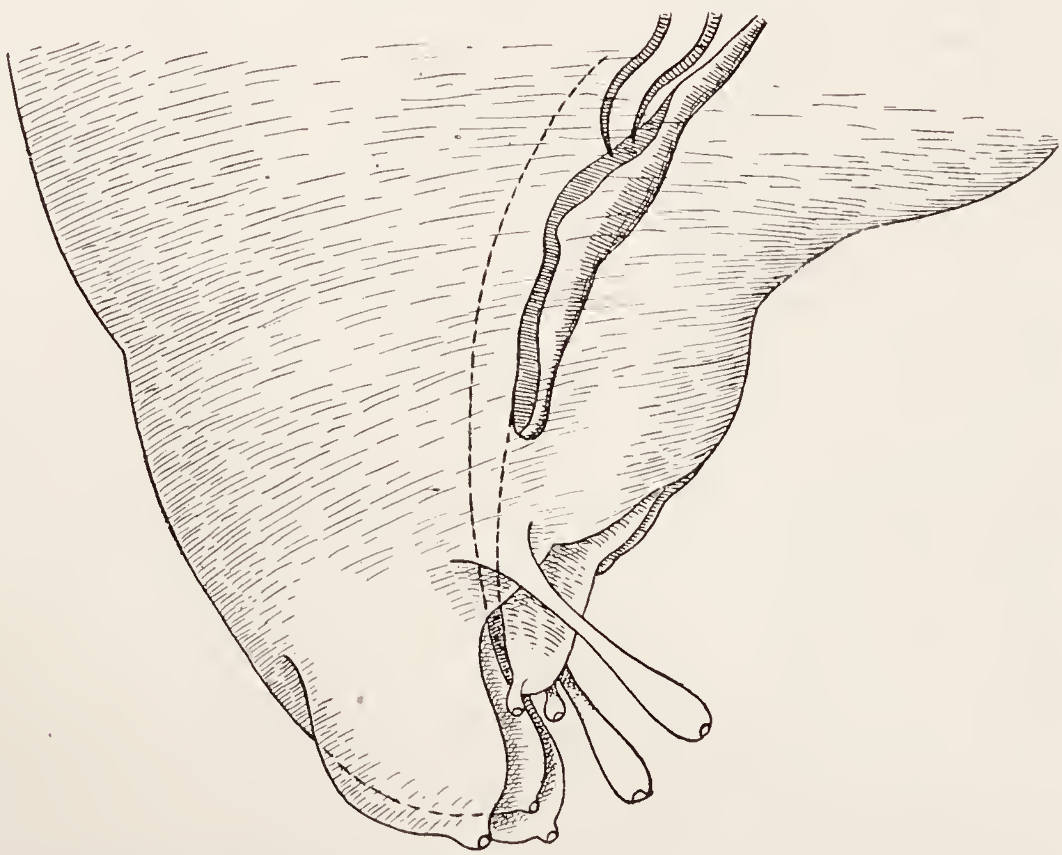


Fig. 47. *Crassisoma urosubulatum.* Male genital cone and appendages. Lateral view. Enlarged. From Alessandrini, 1909.

papilla external to the base of each of the 2 lateral of the 3 papillae; 2 shorter clavate papillae originate on a mammillate base ventral to the cloacal aperture.

Female 6.5 to 7.54 mm. long by 460 to 520μ wide. Esophagus about 752μ long, or 10/77 of total body length. Vulva depressed, situated in the posterior half of the body, 9/14 of the distance from the anterior end; the vulva is transverse, forming an elongate figure 8. Vagina short, passing perpendicular to the body wall into the muscular ovejectors, which are divergent. Anus 227μ from the tip of the tail. Tail (Fig. 48) ends in a transparent mucronation 35μ long, slightly curved dorsal. Eggs (Fig. 49) 52μ long by 35 to 36μ wide.

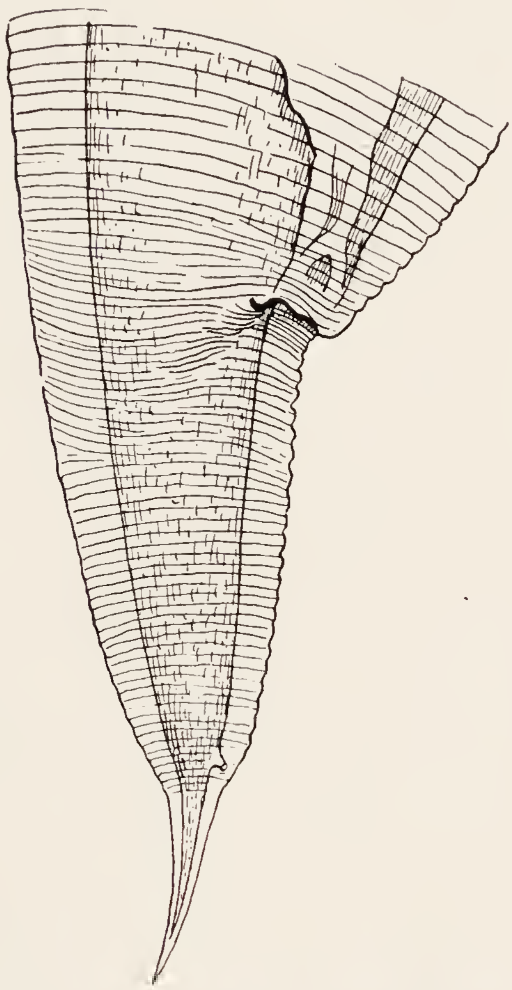


Fig. 48. *Crassisoma urosubulatum.* Female tail. Lateral view. Enlarged. From Alessandrini, 1909.

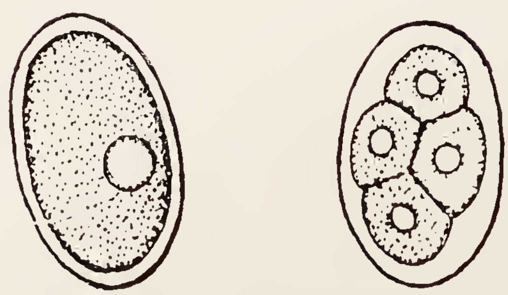


Fig. 49. *Crassisoma urosubulatum.* Eggs. Enlarged. From Alessandrini, 1909.

Life history.—Unknown. The evident relationships of this worm suggest that the life history is similar to those of hookworms. We may surmise that infection takes place both by way of the mouth and by way of the skin.

Distribution.—United States, France, Germany, Austria, Italy, Gold Coast and Argentine. This worm has been reported by Ransom as collected by him at Bethesda, Maryland, and by Dr. F. L. Kilborne at Washington, D. C. It has also been reported by Cram as collected by Raffensperger at Chicago.

Pathology.—Alessandrini notes that the worms attach to the mucosa, causing punctiform hemorrhages in association with an intense catarrhal condition of the intestine. He believes the parasite capable of causing anemia, precisely as hookworms do. The relationships, morphology and habits of the parasite indicate that its pathology is substantially identical with that of hookworms in general. Alessandrini reports the parasite as present in very large numbers in 1 case and counted 200 in another animal.

Treatment.—Unknown. The efficacy of carbon tetrachloride against blood-sucking strongyles in general, and against hookworms in particular, would warrant tests of this substance for the removal of these worms. Carbon tetrachlorid, at a dose rate of 1 ounce in 3 ounces of castor oil for swine weighing 100 pounds, is fairly well tolerated by swine. A large dose of Epsom salts might be better than castor oil. Chenopodium, at a dose rate of 1 fluid dram in 2 fluid ounces of castor oil, is also indicated.

Prophylaxis.—Unknown. Measures similar to those recommended in the case of other hookworms are indicated. Sanitation is important.

At the present time and judging from the available literature, the relationship of this worm to *Globocephalus longemucronatus* is somewhat confused and uncertain. The worms which von Linstow and Railliet have regarded as identical with *G. longemucronatus* actually agree with the description of *C. urosubulatum*. If Molin's description of *G. longemucronatus* is accepted, then we are dealing with 2 distinct species. But there is a possibility that Molin's description is inaccurate, and that *G. longemucronatus* is identical with *C. urosubulatum*, in which case the latter name becomes a synonym of the former. The descriptions of the heads do not agree, but the structure of the bursa, spicules and gubernaculum, as figured, is surprisingly alike for both species. Lane thinks Alessandrini has combined the head of one species with the tail of another. If this confusion has occurred, the present writer is inclined to think Molin confused them, as American material agrees with Alessandrini's description. Railliet, Henry and Joyeux regard the two species as identical. At present it seems advisable to retain both names, keeping in mind the possible identity of these species, especially as Lane has described as *G. connorfilii* a second species of *Globocephalus*.

CRASSISOMA SAMOENSE Lane, 1922

The Samoan swine hookworm

Synonym.—*Raillietostrongylus samoensis* (Lane, 1922) Lane, 1923

Hosts.—Swine.

Location.—Small intestine.

Morphology.—*Crassisoma*: Stout, short worms. Oral aperture (Fig. 50) dorso-subterminal and armed with a pair of rudimentary ventral semilunar plates. Oral capsule (Fig. 51) nearly globular and provided with a pair of large basal subventral teeth, each irregularly quadrilateral in shape, attached by its ventral border and prominently pointed at its free angles. Dorsal esophageal gland or dorsal gutter opens inconspicuously into oral capsule in middorsal line, a little more than half the distance from the base of the capsule.

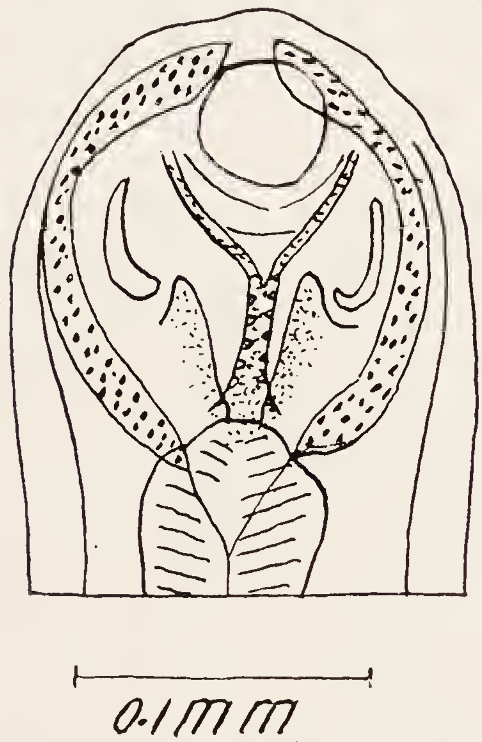


Fig. 50. *Crassisoma samoense.* Head. Dorsal view. From Lane, 1923.

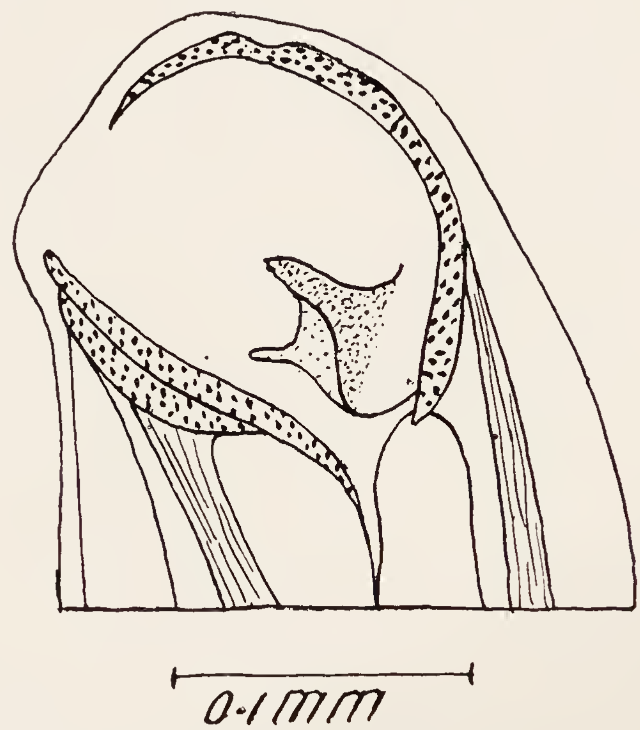


Fig. 51. *Crassisoma samoense.* Head. Lateral view. From Lane, 1923.

Male 4.5 to 5.27 mm. long by 290μ wide. Dorsal rays (Fig. 52) united for about seven-eighths of their length; free portion divides much as in *Globocephalus connorfilii* (i. e., after separation each ray branches almost immediately into an external thin branch and an internal thick branch, the latter again dividing.) The externo-dorsal rays originate at the base of the united dorsal rays. Lateral rays have a common base, are equidistant near the bursal edge, and are of about the same thickness (Fig. 53). Ventral rays fused for ventral half and apposed for the rest of their length. Prebursal papillae small. Spicules (Fig. 54) equal and similar, 380 to 400μ long, giving the appearance of a lyre near their points when viewed dorse-ventrally. Gubernaculum 55μ long, diamond-shaped in dorso-ventral view.

Female 5.25 to 5.5 mm. long by 350μ wide. Distance from anterior end to cervical papillae, 420 to 450μ, about 5/9 of esophagus length

from the anterior end of esophagus; distance to nerve-ring, 400μ; to excretory pore, 425μ; to posterior end of esophagus, 750μ. Tail mu-

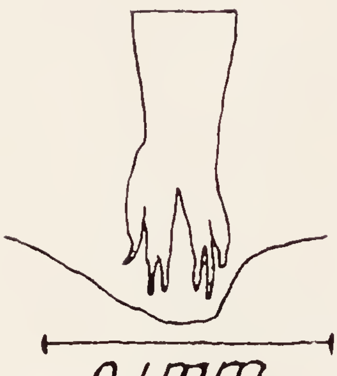


Fig. 52. *Crassisoma samoense.* Termination of dorsal ray. From Lane, 1923.

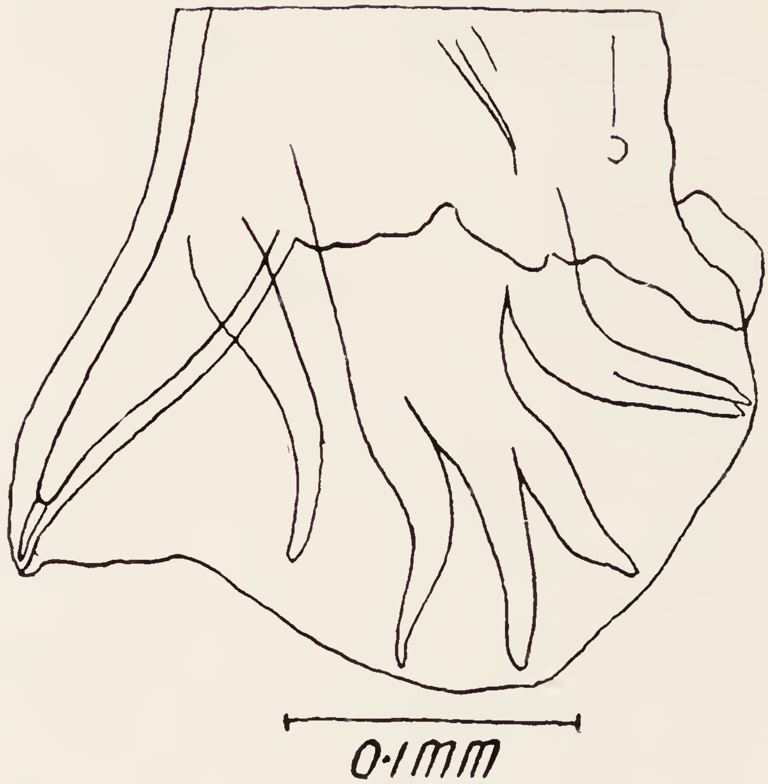


Fig. 53. *Crassisoma samoense.* Bursa. Lateral view. From Lane, 1923.

Fig. 54. *Crassisoma samoense.* Gubernaculum and tips of spicules. From Lane, 1923.

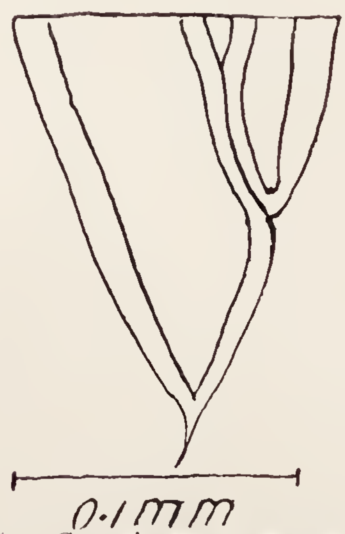


Fig. 55. *Crassisoma samoense.* Female tail. From Lane, 1923.

cronate (Fig. 55). Anus 70 to 85μ from tip of tail. Vulva 2 mm. from tip of tail. Eggs in uterus 65 to 70μ long by 40μ wide.

Life history.—Unknown; probably similar to that of *C. urosubulatum* (See page 73).

Distribution.—Samoa (Lane's first paper gives "Pacific" for this).

Pathology, treatment, etc.—Unknown. Probably similar to *C. urosubulatum* (See page 74).

ANCYLOSTOMA DUODENALE (Dubini, 1843) Creplin, 1845

The Old World hookworm of man

Synonyms.—*Agchylostoma duodenale* Dubini, 1843; *Dochmius duodenalis* (Dubini, 1843) Leuckart, 1876; *Uncinaria duodenalis* (Dubini, 1843) Railliet, 1885.

Hosts.—Swine, occasionally, dog and cat, rarely, and for the most part by experimental infestation, and man, usually. Brumpt states that it occurs in anthropoid apes, Lane reports it from *Felis tigris*, and Adler reports it from the genet and the civet cat.

Location.—Small intestine, especially duodenum.

Morphology.—*Ancylostoma*: Elongate cylindrical worms, attenuated anteriorly in both sexes. Head bent, the mouth opening antero-dorsally. Buccal cavity armed with 2 pairs of ventral teeth curved like hooks and 1 pair of dorsal teeth directed forward (Fig. 56); closely

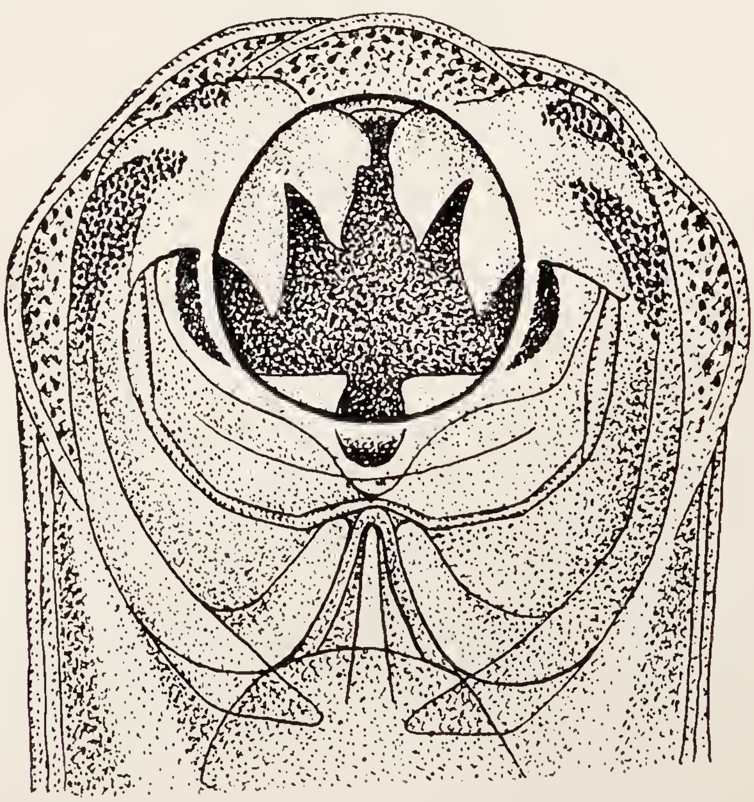


Fig. 56. *Ancylostoma duodenale*. Head. Dorsal view, showing buccal structures. x 200, approximately. From Stephens, 1916, after Looss.

applied about the midline there is a pair of chitinous pharyngeal plates in the floor of the buccal cavity in a latero-ventral position, one at each

side and close to the base of the outer ventral hook; dorsal rib does not project into the buccal cavity. A pair of cephalic glands extend the length of the esophagus and open at the base of the anterior ventral hook. Cervical papillae present.

Male 8 to 11 mm. long by 400 to 500μ wide. Caudal bursa with small dorsal lobe and prominent lateral lobes united by a ventral lobe (Fig. 57). Dorsal ray of bursa divides at a point two-thirds of its length from the base, each branch terminating in 3 digitations. Other rays as given in generic diagnosis. Spicules about 2 mm. long, slender and terminating in fine points, not barbed.

Female 10 to 18 mm. long by 700μ to 1 mm. wide. Vulva at or near union of middle and posterior thirds of body length. Tail conical. Eggs ellipsoidal, 52 to 60μ long by 32μ wide (Fig. 58).

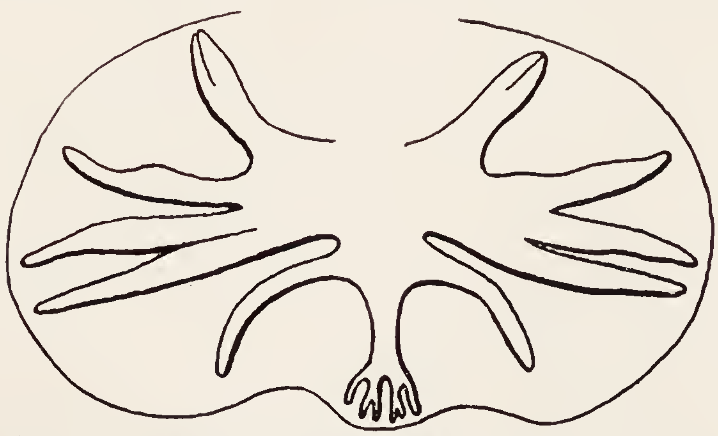


Fig. 57. *Ancylostoma duodenale.* Bursa. Enlarged. From Railliet, 1893.

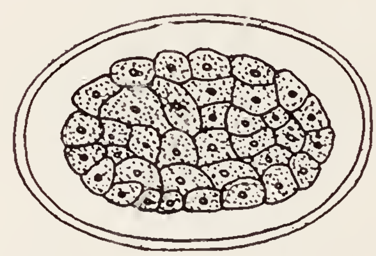


Fig. 58. *Ancylostoma duodenale.* Egg. Enlarged. From Ward, 1907, after Parona and Grassi.

Life history.—Eggs produced by the female worms pass in the feces and hatch. The larvae develop by growth and successive molts to infective third-stage larvae, capable of infecting susceptible hosts either by mouth or through the skin. These larvae enter blood vessels in the digestive tract or under the skin, get to the lungs, ascend the trachea, and are swallowed, developing to adults in the small intestine.

Distribution.—Cosmopolitan for tropical and temperate climates. This worm is seldom found in the United States, the common hookworm of man in this country being *Necator americanus* (See page 82). The records from swine are from the Ellice Islands (O'Connor) and Australia (Legg and Rheuben; Gordon). On re-examination of these specimens Darling finds that some of the worms are *A. duodenale* and some *N. americanus*.

Pathology.—Similar to that of *N. americanus* (See page 82), so far as man is concerned. The pathology in the case of swine needs further study.

Treatment.—Similar, in the case of man, for *N. americanus* (carbon tetrachlorid and chenopodium). Nothing is known about the treatment of swine for infestations with this worm, but presumably the treatment recommended for *Crassisoma urosubulatum* (See page 74) would be worth trying if the extent of infestation warranted it.

Prophylaxis.—Similar to that for *C. urosubulatum*, except that human feces may be regarded as an important carrier of infection. O'Connor has reported this worm from swine in the Ellice Islands. Legg and Rheuben state that they find it relatively common in swine in Queensland, Australia.

NECATOR SUILLUS Ackert and Payne, 1922

The American swine hookworm

Synonyms.—Gordon does not regard the specific characters reported for this species as valid and regards it as *N. americanus* (See page 83).

Hosts.—Swine.

Location.—Small intestine, more often in ileum and jejunum than in duodenum.

Morphology.—*Necator*: Buccal capsule (Figs. 59 and 60) much

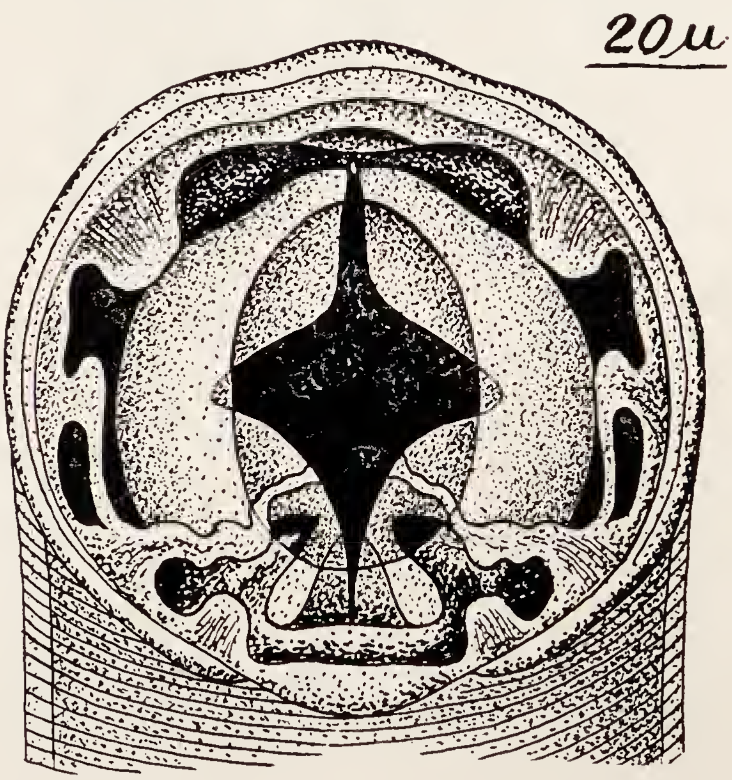


Fig. 59. *Necator suillus*. Head. Dorsal view. From Ackert and Payne, 1923.

smaller proportionately than that of *N. americanus;* the dorsal flexion of the neck less pronounced; inner edges of ventral plates more

rounded and less angular; lateral lancets broadly wedge-shaped in profile and not cusp-shaped as in *N. americanus;* ventral lancet slender, dagger-shaped in lateral view, and pointing toward the base of the dorsal tooth, instead of wider and pointing toward the tip of the dorsal tooth as in *N. americanus.* Esophagus comparatively short and slender.

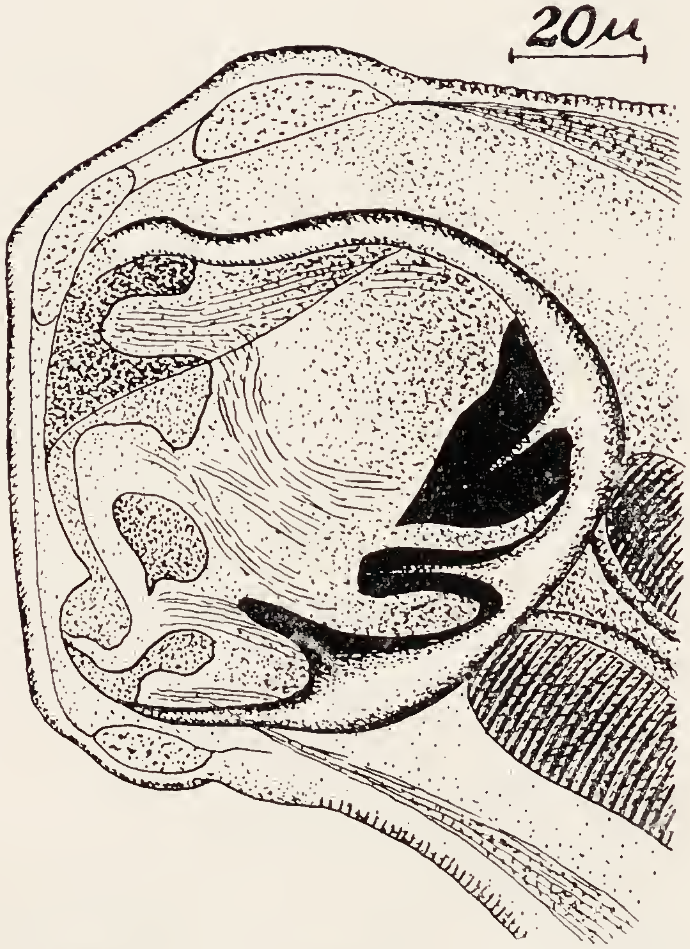


Fig. 60. *Necator suillus.* Head. Lateral view. From Ackert and Payne, 1923.

Male 5.25 to 8.75 mm. long by 90 to 246μ wide. Buccal capsule 57 to 75μ long dorso-ventrally by 58 to 77μ wide. Bursa (Fig. 61) funnel-shaped. Dorsal rays (Fig. 62) short and comparatively stout, with long, slender terminal branches, the rays shorter but their terminal branches longer than those of *N. americanus.* Externo-dorsal ray short and stout. Postero-lateral, medio-lateral and externo-lateral rays short and slender. Latero-ventral and ventro-ventral rays stout. Spicules (Fig. 63) 423 to 471μ long, barbed at the tip.

Female 7.3 to 12.5 mm. long by 295 to 382μ wide. Buccal capsule 68 to 89μ long by 67 to 94μ wide. Vulva slightly anterior to equator of body. Eggs 56 to 66μ long by 35 to 40μ wide, segmenting when deposited.

Life history.—Apparently very similar in a general way to that of *Ancylostoma duodenale* (See page 78). Ackert and Payne report that at least 6 weeks is necessary for worms to develop to maturity. The

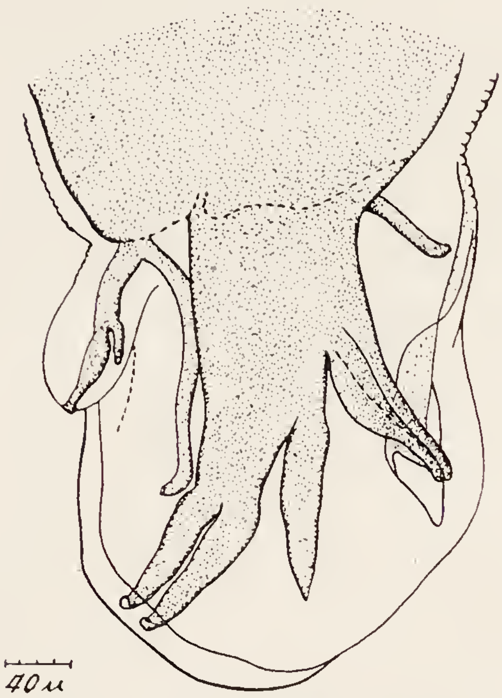


Fig. 61. *Necator suillus.* Bursa. Lateral view. From Ackert and Payne, 1923.

Fig. 62. *Necator suillus.* Dorsal ray of bursa. From Ackert and Payne, 1923.

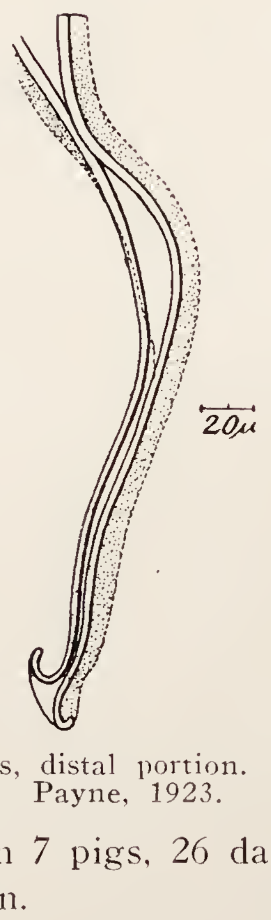


Fig. 63. *Necator suillus.* Spicules, distal portion. Lateral view. From Ackert and Payne, 1923.

occurrence of adult worms in 7 pigs, 26 days old they regard as probably due to prenatal infection.

Distribution.—Island of Trinidad, British West Indies, and (?) Brazil. Over half of the swine in the Island of Trinidad appear to harbor this parasite, according to Ackert and Payne.

Pathology, treatment, etc.—Probably similar to that for *Crassisoma ursubulatum* (See page 74). Ackert and Payne report that the chief effects are muscular deficiency, thickened intestinal mucosa, hemorrhagic wounds of the mucosa, and swollen abdomens.

Gordon has reported the finding in 10 of 15 pigs examined in Brazil of a *Necator* which he regards as probably *N. americanus.* His description corresponds rather closely with that of *N. suillus;* its salient features are: Male length, 4.5 to 6.5 mm.; female length, 5.5 to 8.2 mm.; male width, 230μ; female width, 270μ; male spicule length, 480μ. The occurrence of the same species in pigs in Brazil and in the Island of Trinidad might reasonably be expected. Ackert and Payne were unable to infect young pigs with *N. americanus,* though the larvae produced typical cutaneous lesions. Albiston says Darling has found Australian material reported by Legg and Rheuben as *A. duodenale* to contain *N. americanus* also.

NECATOR AMERICANUS (Stiles, 1902) Stiles, 1906

The American human hookworm

Synonyms.—See Parasites of Dogs.

Hosts.—Pig and dog, rarely; man, usually; apes and rabbits (young stages from experimental infections). Baylis and Lane report it from *Rhinoceros bicornis.* The specimens reported from the pig and the rhinoceros may prove on further study to be *N. suillus* (page 79), if that species proves to be valid.

Location.—Small intestine and, rarely, stomach.

Morphology.—See Parasites of Dogs. The buccal capsule and bursa very similar to that of *N. suillus.* The male is 7 to 9 mm. long and the female 9 to 11 mm. long.

Life history.—Similar to that of *Ancylostoma duodenale* (See page 78).

Distribution.—See Parasites of Dogs. Reported from pig in Brazil and Australia.

Pathology, treatment, etc.—See Parasites of Dogs.

This species was reported from pigs in Brazil by Gordon (tentative determination) and Albiston states that material reported from pigs in Australia by Legg and Rheuben as *Ancylostoma duodenale* was

re-examined by Darling and found to include specimens of *N. americanus* as well as *A. duodenale*. These findings must be reviewed in connection with the report of a distinct species, *N. suillus*, in swine. In such a review Gordon comes to the conclusion that *N. suillus* is identical with *N. americanus* and that the specific characters reported for *N. suillus* are not valid. Gordon tried to infect pigs with *N. americanus* and found what appeared to be hookworm eggs in the feces; he did not find the worms postmortem. Ackert and Payne could not infect swine with *N. americanus*, nor could Goodey. Goodey thinks the eggs found by Gordon may have been those of *Œsophagostomum dentatum*.

UNCINARIA STENOCEPHALA (Railliet, 1884) Railliet, 1885

The narrow-headed hookworm of dogs

Synonyms.—See Parasites of Dogs.

Hosts.—Swine, rarely; dog and cat, usually. Ransom has reported the occurrence of 3 specimens of this worm from the stomach of a pig in Canada.

Location.—Stomach of swine; small intestine of dogs and cats.

Morphology.—See Parasites of Dogs. The buccal capsule has a rounded cutting plate on each side of the ventral wall. The male is 6 to 8 mm. long and the female 8 to 11 mm. long.

Life history.—Similar to that of *Ancylostoma duodenale* (page 78).

Pathology.—See Parasites of Dogs. Of no importance for swine.

Treatment and prophylaxis.—Of no importance for swine.

BUNOSTOMUM TRIGONOCEPHALUM (Rudolphi, 1808) Railliet, 1902

The common sheep hookworm

Synonyms.—See Parasites of Sheep.

Hosts.—Swine, rarely; sheep and goat, usually. Ransom states that there are specimens of this worm, labeled as collected from the pig, in the collection of the Federal Bureau of Animal Industry. There is a possibility, in cases of this kind, that the animal in which the worms were found had recently eaten the intestine of the usual host with its worm content.

Location.—Small intestine in usual hosts.

Morphology.—See Parasites of Sheep. The buccal capsule has a long dorsal tooth and 2 short ventral teeth. The male is 1.2 to 1.7 cm. long, the female 1.9 to 2.6 cm. long.

Life history.—See Parasites of Sheep.

Pathology, treatment, etc.—See Parasites of Sheep. Of no importance for swine.

STEPHANURUS DENTATUS Diesing, 1839

The kidney worm of swine

Synonyms.—*Sclerostoma pinguicola* Verrill, 1870; *Stephanurus nattereri* Cobbold, 1879; *Sclerostomum renium* Drabble, 1922.

Hosts.—Swine and, very rarely, cattle.

Location.—In the abdominal viscera, especially the leaf lard or kidney fat, the liver (including the portal vein and bile ducts), and the lungs, pleural cavity, under the parietal pleura, lumbar muscles, spleen and spinal canal, in swine; in liver of cattle (reported by Hall).

Morphology.—*Stephanurus*: Thick, mottled worms (Fig. 64). Buccal capsule (Fig. 65) suborbicular, its aperture provided with 35 to 40, according to Taylor, or 30 to 34, according to Skrjabin, or about 50, according to Daubney, leaf-like elements forming a mouth-collar, and surrounded by a plaque, in the indentations of which lie 2 lateral papillae (amphids) and 4 submedian papillae; at the base of the buccal capsule are 10 nodules, or according to Daubney, 6 teeth which may be bicuspid or tricuspid. Cuticle transversely striated at 3 to 9μ intervals. Esophagus Indian-club-shaped. Intestine convoluted, much longer than the body of the worm (Taylor says five times as long).

Male 2.0 to 3.7 cm. long, or 4 cm., according to Drabble, by 1.25 mm. wide. The bursa (Fig. 66) is inconspicuous and almost rectangular, forming on each side one lobule sustained by the ventral rays, one sustained by the lateral rays, and one sustained by the dorsal ray system. The ventral rays have a very thick common trunk, only incised a short distance to form the short, thick ventral rays; similarly, the lateral rays have a very thick trunk, incised to form the short, thick lateral rays, widely remote from the ventral rays; the externo-dorsal ray is short and well separated from the lateral rays and the dorsal rays; there are 2 dorsal rays, each consisting of a comparatively short, thick trunk incised to form 3 branches, according to Taylor, or 2 branches according to Skrjabin. Daubney notes that these branches may be bifurcate or trifurcate. Two saber-form spicules usually equal and 800 to 900μ long, but sometimes unequal, the left 930μ to 1 mm. long, the right 660 to 900μ long, with a tubular portion 28μ wide and a lamella as wide, or wider, with transverse striations giving a comb-like or fringelike appearance.

Female 2.5 to 4.5 cm. long, or 5.4 cm., according to Drabble, by 2 mm. wide. The tail is curved ventrally and is obtuse, except for a conical tip. The caudal extremity is provided with lateral cuticular

Fig. 64. *Stephanurus dentatus.* Entire worms. Lateral view. *a*, male, natural size; *b*, female, natural size; at right, female, enlarged. From Taylor, 1900.

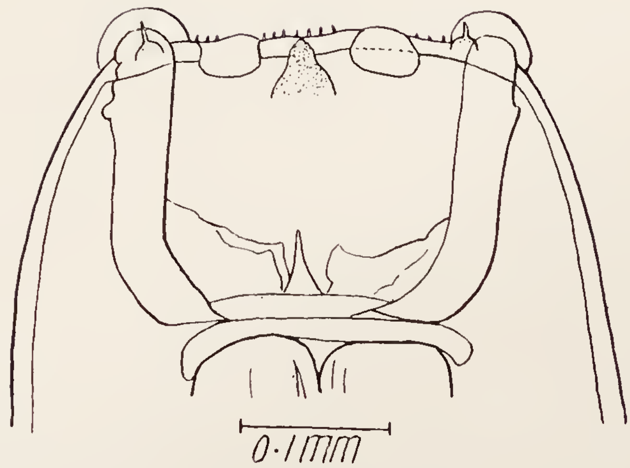


Fig. 65. *Stephanurus dentatus.* Head. Lateral view. From Daubney, 1923.

alae. Anus 590 to 640μ from the tip of the tail. Vulva 993μ to 1.36 mm. anterior of the anus. Eggs 90 to 120μ long by 55 to 65μ wide, and thin-shelled (Fig. 67), segmenting when deposited.

Larva, first stage, 420 to 500μ wide, and rhabditiform. The cylindrical pharynx, 10 to 14μ long, is armed with 2 small, slender teeth. The esophagus has 2 bulbs; the second bulb is provided with a tridentate armature. Body widens through the region of the bulb and then thins gradually to a long, rather blunt tail. After about 5 days, there is

a molt and the **second stage larva,** which is strongyloidiform, appears. This is 600μ long by 40μ wide, including its surrounding sheath.

Life history.—This life history was ascertained by Bernard and Bauche. In their normal location, in the kidney fat or tissue, the worm has a fistulous connection with the kidney or ureters. A male and a female are commonly present in the same cyst. The eggs pass in the urine and under favorable conditions of temperature and moisture, rhabditiform embryos hatch after 48 hours and up to the fourth day. Within 5 days this larva develops to a second stage larva after a molt. The infective larvae enter their host either by way of the mouth or the skin. Apparently the cutaneous route is the normal one, as such larvae

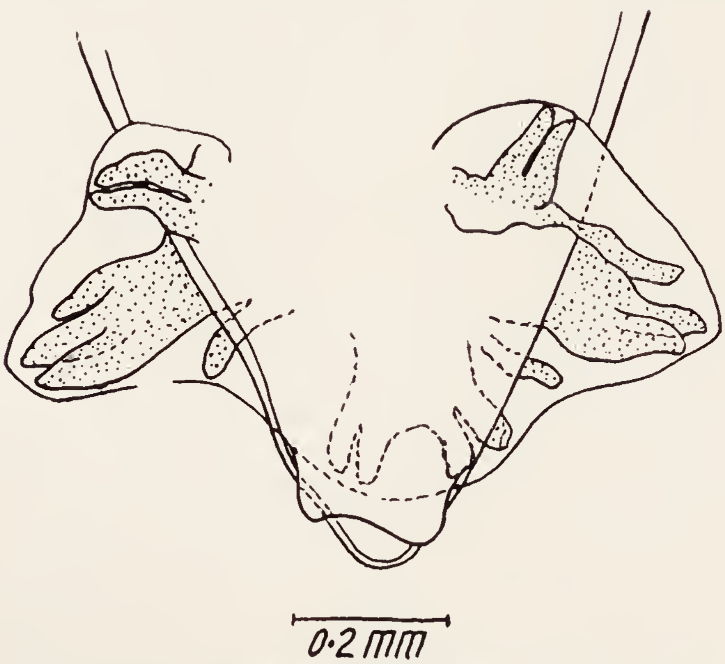


Fig. 66. *Stephanurus dentatus.* Male tail. Ventral view. From Daubney, 1923.

Fig. 67. *Stephanurus dentatus.* Egg containing embryo. Enlarged. From Taylor, 1900.

make their way to the kidney region, from which position the eggs may return to the outside world. Those larvae which enter by way of the mouth make their way to the liver, which must be regarded as an abnormal and unsatisfactory location, from the standpoint of the worm, as the worms do not produce eggs. Even if eggs were produced, they would remain in the liver with but little chance of making their way to the exterior of the body. Worms in the lungs do not become mature.

Distribution.—United States, British West Indies, Guam, South America (Brazil, Uruguay and Argentina), Australia, Java, Sumatra, Philippines, Indo-China and Africa (Dahomey and Belgian and French Congo).

Pathology.—The fat about the upper part of the ureters is usually much thickened, and in massive infestation the entire kidney region is

involved in a mammillated, adipose tumor formation. In this the worms lie in cavities full of greenish fluid, which connect with the ureters by fistulae. This worm has been regarded as in some way capable of causing or spreading hog cholera, an idea which has at this time no evidence to support it. It has also been very commonly regarded by veterinarians, farmers and stock raisers in the Southern United States as the cause of a weakness or paralysis of the hind quarters in swine, and there is a considerable amount of evidence and theoretical support in favor of this belief. So far as may be judged from incidental evidence, which is not well compiled or digested at this time, this paralysis of the hind quarters is quite prevalent in those sections of the southern United States where this worm is most prevalent. It is also theoretically possible that the fistulous lesions associated with these worms in the kidney region may give rise to the lumbar reflexes of kidney disease, and that toxins in the secretions or excretions of the worms are absorbed with effects which are most pronounced in the lumbar region. This subject needs further investigation, but the belief can hardly be dismissed off hand. Occlusion of the portal vein by the worms may result in fatal colic. Drabble states that the worms may cause hydronephrosis and that the kidney may be reduced to a fibrous sac containing a few nodules. He also reports a cystitis.

The worms in the liver give rise to abscess formation, with bacterial involvement, and here again is the likelihood of toxic absorption in addition to the evident mechanical damage to an important gland. In Dahomey, where liver infestation is as common as kidney infestation, Pecaud regards the worm as the cause of a profound cachexia. Loss of appetite, emaciation and swollen abdomen are the usual symptoms. Gonzalez and Lago find it the most serious cause of deaths among pigs in some parts of the Philippines.

From the standpoint of meat inspection, the damage to carcasses, from which wormy livers and leaf lard must be commonly discarded, makes a considerable aggregate for the entire area over which this worm is found, making the worm of considerable interest from this standpoint alone.

Cutaneous lesions in the form of papules are quite marked after massive infection through the skin, and the nearby glands hypertrophy. Larvae may be found in these glands.

Treatment.—Unknown; the site of infection of this worm puts it beyond the reach of anthelmintics in the present stage of our knowledge.

Prophylaxis.—This is evidently a matter of sanitation. It is essential that swine be kept under cleanly conditions, with especial care in regard to the frequent and thorough cleaning of yards and buildings occupied by them, in order that the eggs passed in the urine may be removed before reaching the infective stage. It is not only necessary to prevent contamination of food and water to prevent entrance into the body by way of the mouth, which results in infestation of the liver, but also to prevent cutaneous infection in the wallows, which results in kidney infestation. Concrete floors and raised slat floors are of value in prevention of infestation.

HYOSTRONGYLUS RUBIDUS (Hassall and Stiles, 1892) Hall, 1921

The red stomach worm of swine

Synonyms.—*Strongylus rubidus* Hassall and Stiles, 1892; *Ostertagia rubida* (Hassall and Stiles, 1892) Travassos, 1918; *Trichostrongylus rubidus* (Hassall and Stiles, 1892) Fiebiger, 1923.

Hosts.—Swine.

Location.—Stomach, especially in fundus.

Morphology.—*Hyostrongylus*: Small, reddish worms. Head slightly inflated. Cuticle finely striated transversely and with 40 to 45 longitudinal striations. Esophagus divided indistinctly into 2 portions. Intestine greyish-black, wound spirally with the genitalia.

Male 5 mm. long by 87 to 128μ wide. Bursa 195μ long by 300μ wide, the dorsal lobe very small, but quite distinct (Fig. 68). The latero-ventral ray is larger than the ventro-ventral ray and its tip turns back toward the tip of the ventro-ventral. The externo-lateral and medio-lateral rays diverge slightly, the postero-lateral ray diverging more widely from the medio-lateral. The externo-dorsal ray lies about midway between the postero-lateral rays and the dorsal ray. The dorsal ray bifurcates near its tip and has 2 small branches at about two-thirds of the distance from the base. Two equal spicules, 130μ long by 20μ wide anteriorly, tapering to a point, with a wavy ridge running the length of the spicule and supporting a curved membranous portion, which terminates in a second point. Posterior of the usual position of the spicules in the body is a narrow gubernaculum, 60μ long. Embracing the gubernaculum is a clear refractile structure, the telamon (Fig. 69), having a central portion shaped like a spur or a wish-bone, with the point of the spur extending anteriorly; from the 2 posterior points of the spur, 2 curved flat plates extend anteriorly and lie in the posterior wall of the cloaca on each side, the curvature of the plates corresponding to that of the cloacal walls (Fig. 69).

Female 8 to 8.5 mm. long by 110μ wide. Tail 68μ long. Vulva 1.3 to 1.5 mm. anterior of the anus; just posterior of the vulva is a

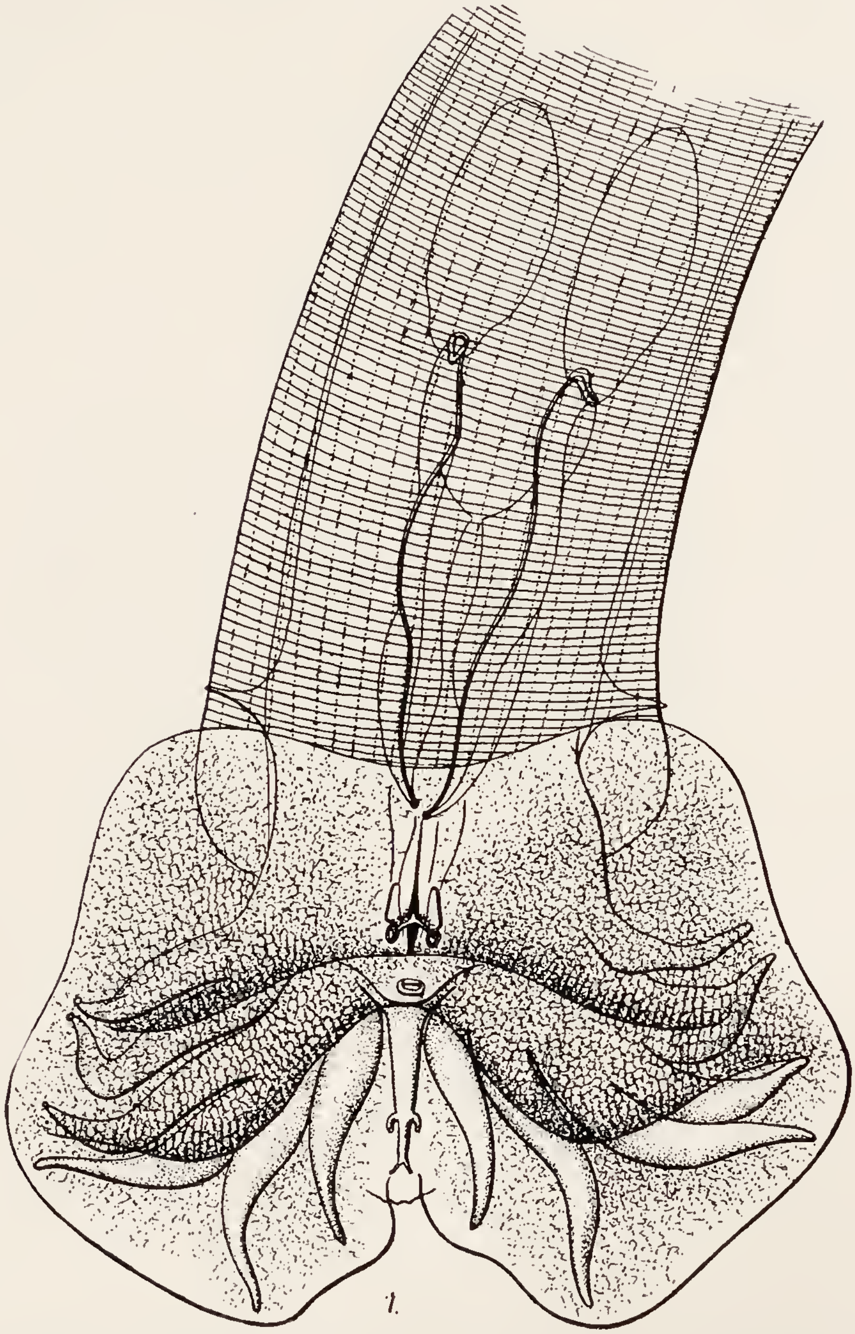


Fig. 68. *Hyostrongylus rubidus.* Male tail. Enlarged. From Hassall and Stiles, 1892.

small, crescentic cuticular fold, 40μ long by 13μ wide. Vagina bottle-shaped and at right angles to body wall. Eggs elliptical, 45μ long by 36μ wide, segmenting when deposited (Fig. 70).

Life history.—Unknown; probably simple and direct, the eggs passing in the feces, hatching, the young worms developing to the infective larvae and being ingested by swine with contaminated food or water, and developing to adults in the stomach of the host.

Distribution.—United States, Germany, Hungary and England.

Pathology.—This worm has been found usually in connection with an excess of thick gastric mucus, suggesting that it occasions a chronic catarrhal condition. Crocker and Biester find females to be most common on the surface of the croupous membrane thus produced, and males most common in the depths of the membrane. Von Oppermann has reported it as the cause of a disease of swine in Germany. It

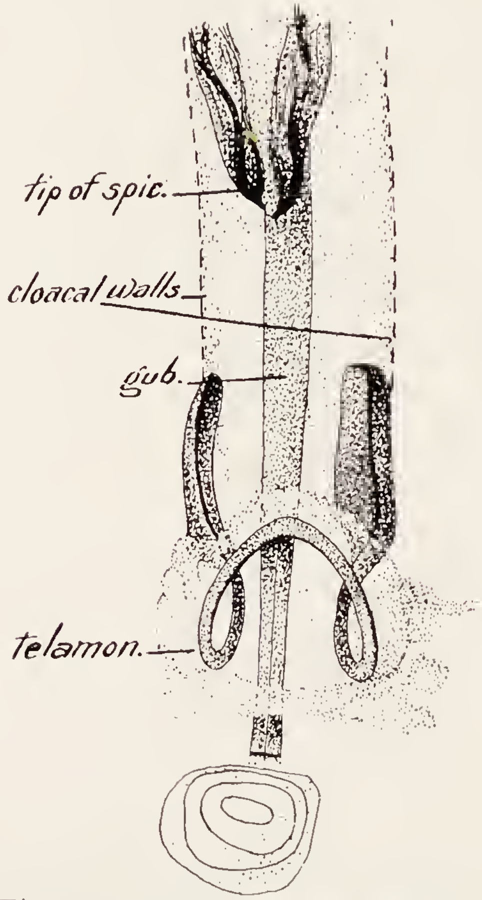


Fig. 69. *Hyostrongylus rubidus.* Cloacal region of male. Enlarged. From Hall, 1921.

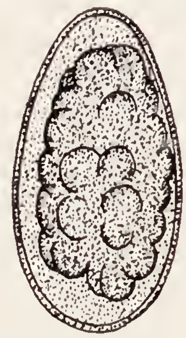


Fig. 70. *Hyostrongylus rubidus.* Egg. x 250. From Marek, 1922.

often occurs in such large numbers as to give the gastric mucosa the appearance of hemorrhage, and it is quite likely that it will be found to cause considerable injury. Oppermann and, later, Crocker and Biester find that the worm produces a heavy catarrhal exudate and small ulcerations of the gastric folds. Oppermann regards the worms as the only essential and primary cause of these lesions, whereas Crocker and Biester consider that a preliminary disease process, such as broncho-pneumonia, even though of slight degree, is an essential antecedent factor in lowering the gastric vitality and tone and thus permitting the worms to exert a pronounced pathological action.

Treatment.—Unknown. Unless the mechanical protection afforded by the excessive mucus associated with the worm inhibits the action of anthelmintics, the removal of this worm from the stomach should pre-

sent no especial difficulties. Carbon tetrachlorid would be worth trying. Oil of chenopodium, in doses of 1 dram for a 100-pound animal might be of value. Turpentine or copper sulphate solution (See page 146) might also be serviceable.

Prophylaxis.—Sanitary measures, the prompt removal and proper disposal of manure, and the maintenance of clean quarters and yards for swine are indicated.

MECISTOCIRRUS DIGITATUS (Linstow, 1906)

Railliet and Henry, 1912

The digitate mecistocirrid

Synonyms.—See Parasites of Cattle.

Hosts.—Swine, sheep, cattle, buffalo and (?) man. Swine and sheep are reported as hosts of this worm by Cameron. The usual hosts are bovines.

Location.—Fourth stomach of ruminants; presumably stomach of swine. Cameron does not specify the location in the case of swine. Since Cameron makes *M. fordi* a synonym of *M. digitatus* it is possible that the reports of that worm are referred to. It is reported from stomach and intestine.

Morphology.—See Parasites of Cattle. Filiform worms, the anterior portion of the body slenderer than the posterior portion. Males 16 to 24 mm. long; females 19 to 29 mm. long.

Life history.—Unknown; probably simple and direct. See Parasites of Cattle.

Distribution.—Ceylon, Sumatra, Formosa, China and India.

Pathology, treatment, etc.—See Parasites of Cattle. Of no known importance for swine.

MECISTOCIRRUS FORDI (Daniels, 1908)

Railliet and Henry, 1912

Ford's mecistocirrid

Synonyms.—*Strongylus fordii* Daniels, 1908.

Hosts.—Swine and also cattle and man.

Location.—Stomach and intestine.

Morphology.—See Parasites of Cattle. Worms very similar to *M. digitatus* (page 91). Males 19 mm. long; females 21 mm. long.

Life history.—Unknown; probably simple and direct. See Parasites of Cattle.

Distribution.—East Indies, Dutch East Indies, India and China.

Pathology, treatment, etc.—See Parasites of Cattle. Of no known importance for swine.

METASTRONGYLUS ELONGATUS (Dujardin, 1845)

Railliet and Henry, 1911

The common lungworm of swine

Synonyms.—*Gordius pulmonalis apri* Ebel, 1777; *Ascaris apri* Gmelin, 1789; *Strongylus suis* Rudolphi, 1809; *Strongylus paradoxus* Mehlis, 1831; *Strongylus elongatus* Dujardin, 1845; *Strongylus longevaginatus* Diesing, 1851; *Metastrongylus paradoxus* (Mehlis, 1831) Molin, 1860; *Metastrongylus apri* (Gmelin, 1789) Railliet and Henry, 1907. *Dictyocaulus viviparus* has been reported from swine, apparently as a result of error.

Hosts.—Swine, commonly, and man, cattle, sheep and goat very rarely. Koch has reported this species from sheep. His figures are evidently *M. elongatus* and the record is probably correct. Sluiter has reported it from the goat and Neveu-Lemaire has reported it from cattle. Hutyra and Marek report it from the deer and roe-deer. It is reported once from the lungs of a child and once from human feces, probably having been eaten in food of some sort and presently passed. *Filaria trachealis* Cobbold, from the trachea and larynx of man, is regarded as this species.

Location.—Bronchi and trachea.

Morphology.—*Metastrongylus*: Slender, white worms (Fig. 71) with truncate head bearing a small mouth (Fig. 72), consisting of a

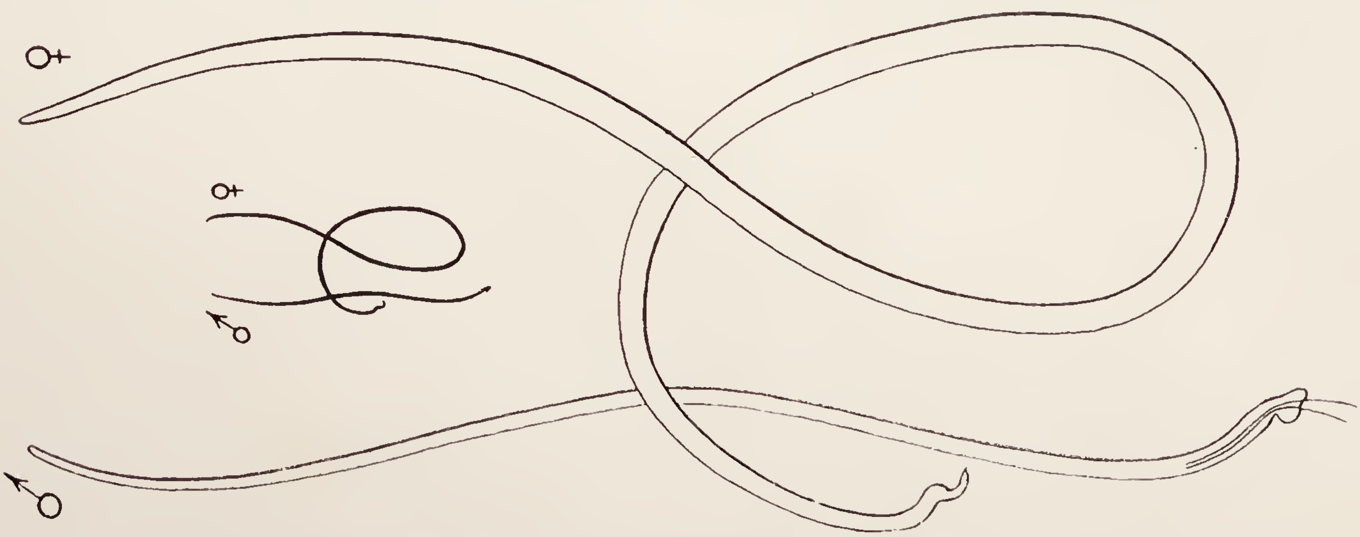


Fig. 71. *Metastrongylus elongatus.* Male and female as indicated. Small figures, natural size; large figures, x 5. From Hall, 1922, after Neveu-Lemaire.

dorso-ventral aperture bounded by 2 lateral lips of 3 lobes each, the median the larger, and surrounded by 6 papillae, of which the lateral are the larger; Gedoelst states that he does not find these papillae.

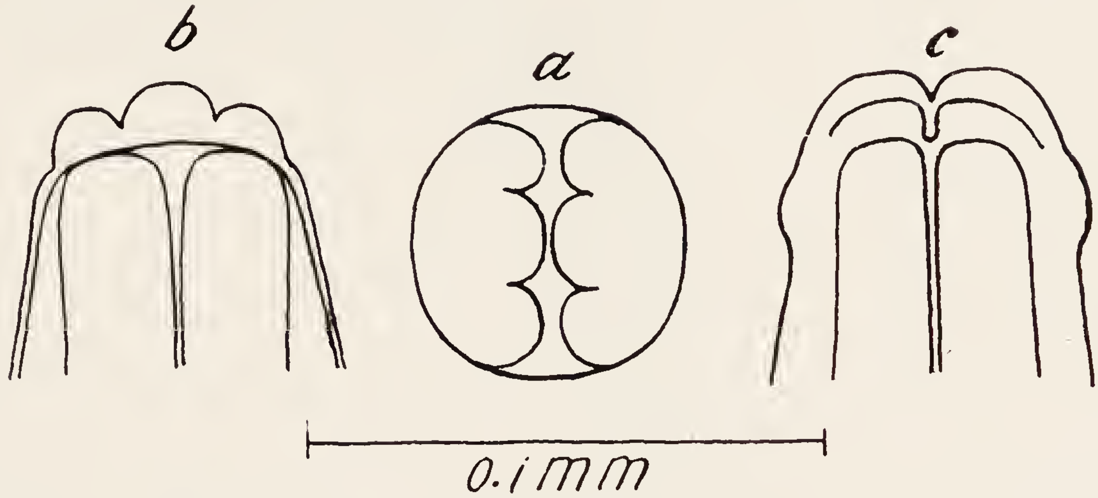


Fig. 72. *Metastrongylus*. Diagrammatic views of head. *a*, viewed from front; *b*, lateral view; *c*, dorso-ventral view. From Gedoelst, 1923.

Male 1.1 to 2.5 cm. long, according to some writers, or 1.6 to 1.82 cm., according to Gedoelst, by 160 to 225μ wide. Esophagus 500μ long. Bursa (Fig. 73) with 2 lobes, difficult to spread out, not prolonged posteriorly, the wall thickened distally, the long axis more or less parallel to that of the body. The dorsal rays and the externo-dorsal rays are narrow; all others are thick. The tip of the latero-ventral ray curves away from the ventro-ventral ray. The tip of the externo-lateral ray ends in a large lobed swelling. The medio-lateral and postero-lateral rays are well developed. The spicules are very long, 4 to 4.2 mm., according to Gedoelst (other writers say 2.5 to 5 mm.), thin and with a striated lateral wing along the entire extent, and each terminates in a single simple hook. No gubernaculum. Genital cone well developed.

Female 2 to 5 cm. long, according to some writers, or 3.9 to 4.2 cm., according to Gedoelst, by 400 to 450μ wide. Esophagus 630μ wide. Intestine commonly shows as a dark line. Tail terminates in a short hornlike process (Figs. 73, 74), and the posterior extremity of the body tends to fold on itself for a distance of 270 to 600μ (Fig. 73). Vulva just in front of the anus, and with a prominent swelling just anterior to it. Common trunk of ovejector (Fig. 74) about 2.3 mm. long. The uteri are convergent. The eggs are said by some writers to be 57 to 100μ long by 39 to 73μ wide; according to Zebrowski they are 50 to 80μ long; according to Gedoelst they are 51 to 54μ long by 33 to 36μ wide. They contain a well developed embryo when deposited. Zebrowski states that the eggs have a thick, gelatinous outer shell and a thin inner membrane.

Embryo 220 to 350μ long by 10 to 12μ wide, granular posteriorly and clear anteriorly and with a bent knob-like tail. Zebrowski says the embryo is 220 to 250μ long, the embryo of *M. brevivaginatus* being larger.

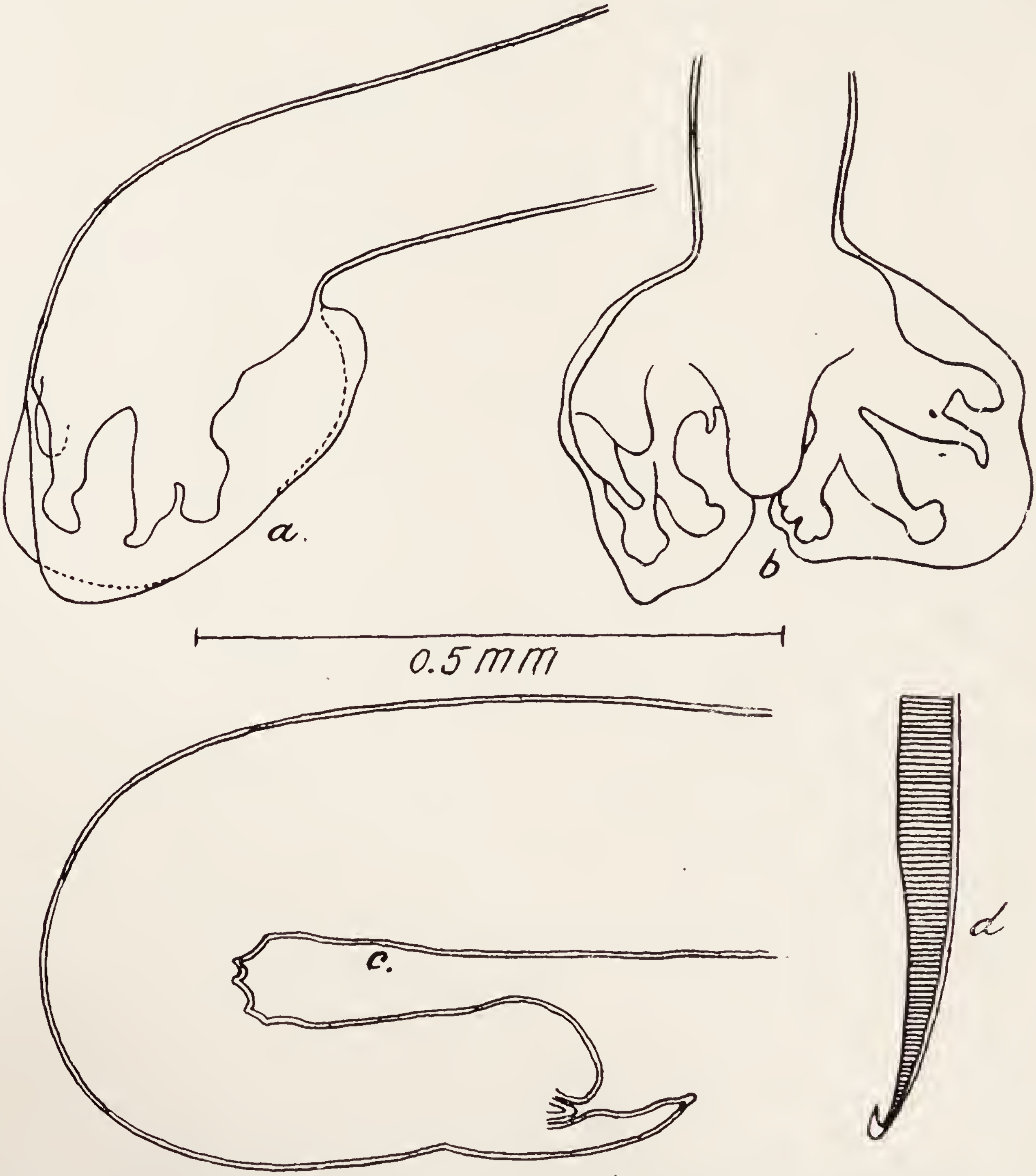


Fig. 73. *Metastrongylus elongatus.* *a*, male tail, lateral view; *b*, male tail, ventral view; *c*, female tail, lateral view; *d*, distal end of spicule. From Gedoelst, 1923.

Life history.—According to Zebrowski, the life history is as follows: The eggs contain active embryos when deposited and these hatch in the lungs. The young larvae can be detected in scrapings from the trachea, the sinuses of the head and the nasal discharges. Presumably they pass out for the most part in the feces. Larvae taken

from the lungs developed normally. Under suitable environmental conditions the larvae (Fig. 75) grow actively for 4 weeks, after which time no growth is observed. The most favorable temperatures are be-

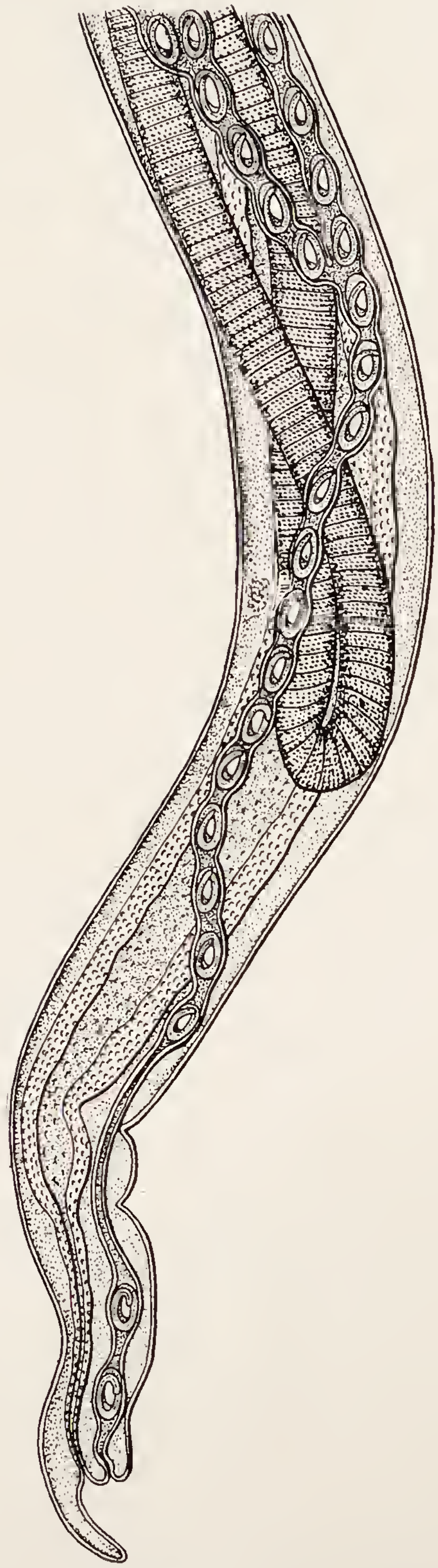


Fig. 74. *Metastrongylus elongatus.* Female tail. Lateral view. x 45. From Neveu-Lemaire, 1918.

tween 35 degrees to 40 degrees C., preferably the upper limits of this range. The moisture requirement is rather exact, too much excluding

the required oxygen supply and too little inhibiting motion and proper metabolism. Damp, porous soils with much organic matter were most satisfactory for culture media, and growing vegetation aided in controlling the moisture requirements. In the course of 4 weeks the digestive tract and sex organs become defined. The spine-like tail in the male larva is much longer than in the female, and the male is slender and active while the female is thick and sluggish. Ovaries can be seen in the latter during the second week. The length increases up

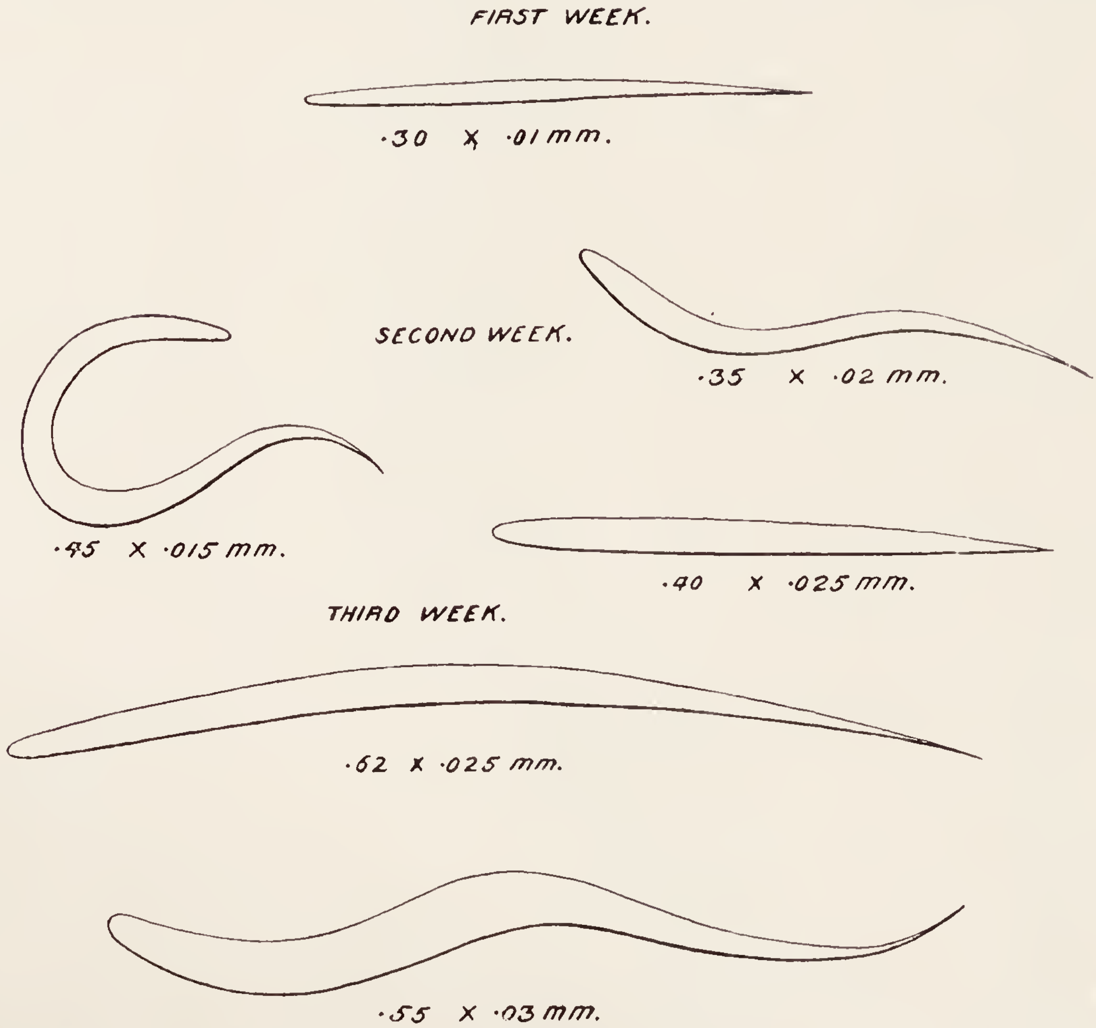


Fig. 75. *Metastrongylus elongatus.* Diagram showing development of larvae in size and outline. From Zebrowski, 1921.

to about 740μ and the width to 55μ. (These figures may apply only to *M. brevivaginatus*, said to be the larger; Zebrowski reports on mixed cultures.) During the next 6 weeks the larvae molt, but the time, number and nature of the molts is not stated by Zebrowski. These larvae may live under laboratory conditions for almost a year. Zebrowski fed 2 rats with well developed larvae at intervals of 1 week for 10 weeks, killing the rats 2 weeks after the last feeding. Both showed verminous lesions in the lungs, the lesions containing numerous dead larvae and molted cuticles, but no live larvae. Three pigs were fed larvae and this

feeding repeated 49 days later. Nineteen days later these pigs were killed. One had generalized verminous bronchitis; another 5 lesions with 96 worms; the third had 6 lesions with 105 worms. Three check animals from the same lot, which had ranged in a hog lot with the experiment animals until 3 months old, all animals being of the same age, were killed at the same time. One had 1 lesion with 8 worms; another 1 lesion with 9 worms; the third 1 lesion with 13 worms. While the checks showed some infestation, the much higher infestation in the animals fed larvae, taken in connection with what is known of the life history of lungworms in other animals, indicates that *M. elongatus* has a simple life history, with direct infection without the necessity of an intermediate host or a free-living generation. It is probable that the worms get to the lungs by way of the blood-stream and there is the possibility of prenatal infection. Zebrowski notes that Dr. Craig has found adult lungworms in pigs 3 weeks old.

Zebrowski finds the worms most abundant in summer and fall, becoming less common in winter, and least common in March.

Distribution.—United States, Porto Rico, Mexico, British West Indies, Yucatan, Argentina, Europe, Belgian Congo, Annam, Japan and Australia.

Pathology.—The disease produced is a verminous bronchitis, characterized by a cough. Heavy infestations sometimes cause pneumonia. Infestation with this worm usually is not fatal, but may kill young animals. The post mortem lesions and other features of the disease are very similar to those of infestation with *Dictyocaulus viviparus,* cattle lungworms, with pneumonia conditions more pronounced. The posterior tip of the lungs commonly shows grey consolidated areas, rather sharply delimited. In old hogs there may be extensive watery tumors containing remnants of dead worms. Small nodules, suggestive of tubercles, may be present. A diffuse pneumonia may result in a dry puffiness of lung tissue. Bacterial complications are not uncommon. Kinsley states that 60 per cent of the lungs of 1000 swine at a Kansas City abattoir contained lungworms in such numbers that the lesions could readily be observed on gross examination. Zebrowski found 780 Indiana swine, or 53.4 per cent, infested with lungworms. In 4 animals showing average infestations he found from 111 to 186 worms, 51 per cent of which were males.

Sparapani says lungworm infestation in swine may be diagnosed by injections, intradermally, of an antigen prepared from the nasal secretions of the animal injected. Dissolve the secretion in an equal amount of physiological saline; add 3 per cent acetic acid, 3 or 4 drops

to 10 cc., to precipitate the mucin; filter to remove mucin; neutralize the filtrate with 3 per cent potash solution; filter through Berkefeld filter. If a few drops of the resultant fluid are injected intradermally, infected animals will show a reaction comparable to the tuberculin reaction.

Treatment.—The treatment which appears to be safest and best is good nursing in connection with abundance of good feed, safe drinking water and shelter. Infected animals should be isolated and taken off infected pastures. Intratracheal injections aside from their uncertain safety and general lack of efficacy, are not suited to swine owing to the amount of fat in the neck region.

Prophylaxis.—So far as the life history of this worm may be surmised from what we know of similar worms, the measures recommended in the case of swine ascarids (See page 60) will be of service in preventing infection with lungworm.

METASTRONGYLUS SALMI Gedoelst, 1923

Salm's swine lungworm

Synonym.—*Metastrongylus elongatus* Salm, 1918, not Railliet and Henry, 1911.

Hosts.—Swine.

Location.—Bronchi.

Morphology.—*Metastrongylus*: Head very similar to that of *M. elongatus* (See page 92).

Male 1.7 to 1.8 cm. long by 225 to 255μ wide. Esophagus 500μ long. Genital cone feebly developed. Caudal bursa small (Fig. 76), its lateral lobes strongly prolonged posteriorly and with the walls strongly thickened in the distal portions. Latero-ventral ray with its extremity bent dorsally. Externo-lateral ray voluminous, terminating in a swelling which is more or less lobulate. Spicules of median length, 2 to 2.1 mm. long, with a striated lateral wing along their entire extent, and terminating in a single hook with a retrorse barb. Gubernaculum absent.

Female 3 to 4.5 cm. long by 320 to 285μ wide. Esophagus 600μ long. Posterior extremity folded at an angle along the ventral surface (Fig. 76) for a distance of 500 to 600μ. Judging from Gedoelst's figures, the swelling anterior to the vulvo-anal region is distinctly smaller than in *M. elongatus*. Vagina, or common trunk of ovejector, of medium length, 1.6 mm. long. Eggs 52.5 to 55.5μ long by 33 to 40μ wide.

Life history.—Probably similar to that of *M. elongatus* (See page 94).

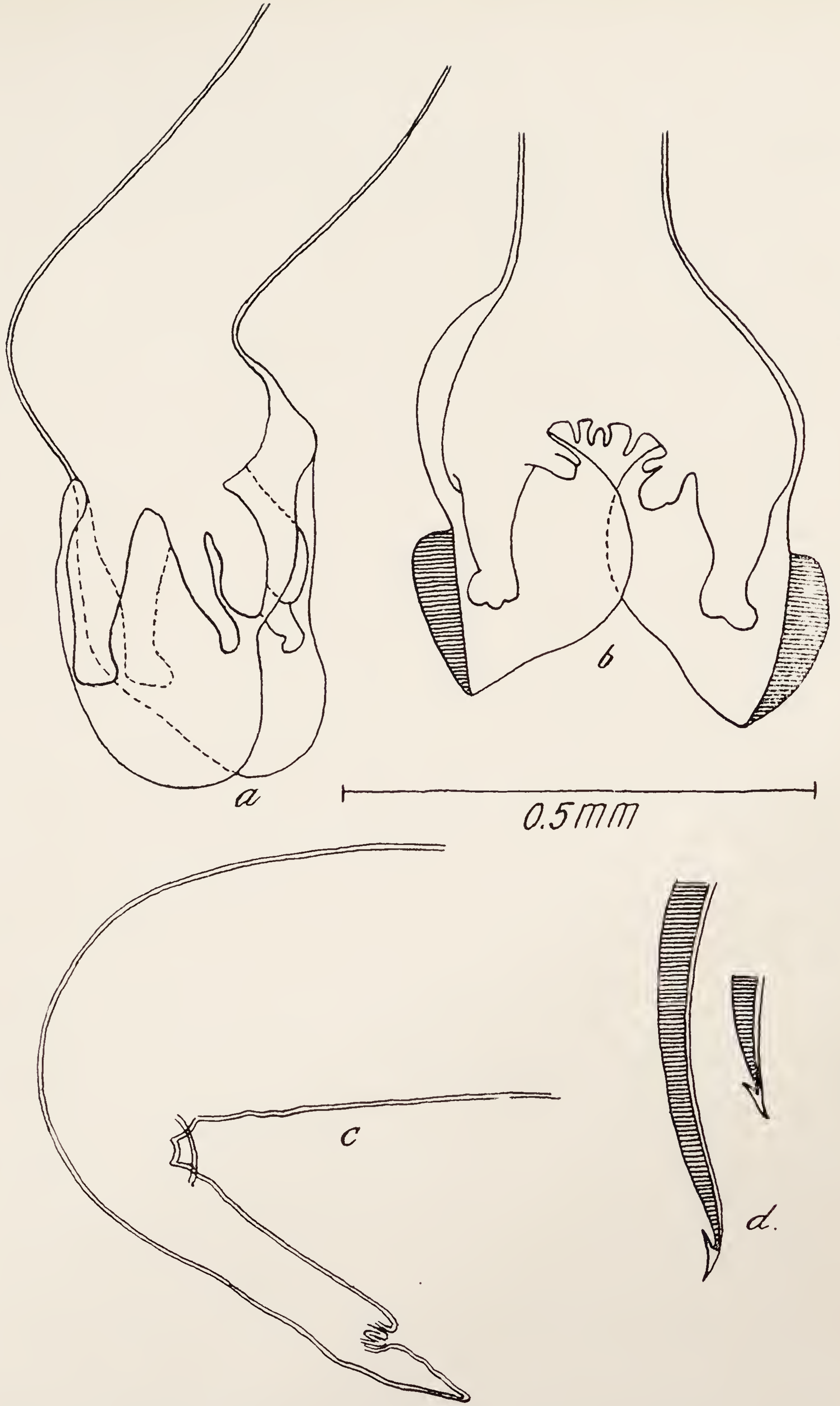


Fig. 76. *Metastrongylus salmi.* *a,* male tail, lateral view; *b,* male tail, dorsal view; *c,* female tail, lateral view; *d,* distal end of spicules. From Gedoelst, 1923.

Distribution.—United States, Belgian Congo and, apparently, Java. Probably widely distributed. Schwartz finds this species in the collection of the Bureau of Animal Industry, part of the material being collected by Foster in 1913 and labeled "Metastrongylus n. sp."

Pathology, treatment, etc.—Probably similar to those for *M. elongatus* (See page 97).

CHOEROSTRONGYLUS BREVIVAGINATUS (Railliet and Henry, 1907) Gedoelst, 1923

The short-vagina lungworm of swine

Synonym.—*Metastrongylus brevivaginatus* Railliet and Henry, 1907.

Host.—Swine.

Location.—In bronchi and trachea.

Morphology.—*Choerostrongylus*: Head very similar to that of *M. elongatus* (See page 92).

Male 1.2 to 2.5 cm. long, according to some writers, or 1.6 to 1.8 mm. long, according to Gedoelst, by 270 to 295μ wide. Esophagus 480 to 500μ long. More opaque, thicker and less tightly coiled than *M. elongatus.* Bursa (Fig. 77) with long axis perpendicular to that of body and its walls not thickened in distal portion. The extremity of the latero-ventral ray is not bent dorsally and terminates in a small swelling. The externo-lateral ray is not swollen at its extremity. Spicules 1.2 to 1.5 mm. long, the striated lateral wing lacking along the anterior fourth, each terminating in a double hook (Fig. 77). Gubernaculum present, 40μ long and gutter-shaped. Genital cone feebly developed.

Female 2 to 5 cm. long. Esophagus 520 to 560μ long. Vulva near anus. A large ventral swelling anterior to the anus has the vulva on its posterior margin, and the inflated cuticle of this swelling forms a vesicle extending from the anterior margin of the swelling to the level of the tip of the tail and far ventral of the swelling itself (Fig. 77). Vagina or common trunk of the ovejector about 480 to 535μ long. Eggs said to be the same size as those of *M. elongatus.* According to Zebrowski, the eggs are 70 to 100μ long by 50 to 80μ wide; according to Gedoelst they are 57 to 63μ long by 39 to 42μ wide. The figures usually given for *M. elongatus* may be composite figures.

Life history.—Similar to that of *M. elongatus,* according to Zebrowski, the corresponding larvae being somewhat larger in *M. brevivaginatus.*

Distribution.—United States, British West Indies, Argentine, Europe, Annam and Belgian Congo. In Indiana hogs Zebrowski found this species about as common as *M. elongatus* (Page 97).

Pathology, treatment, etc.—See *M. elongatus*, page 97).

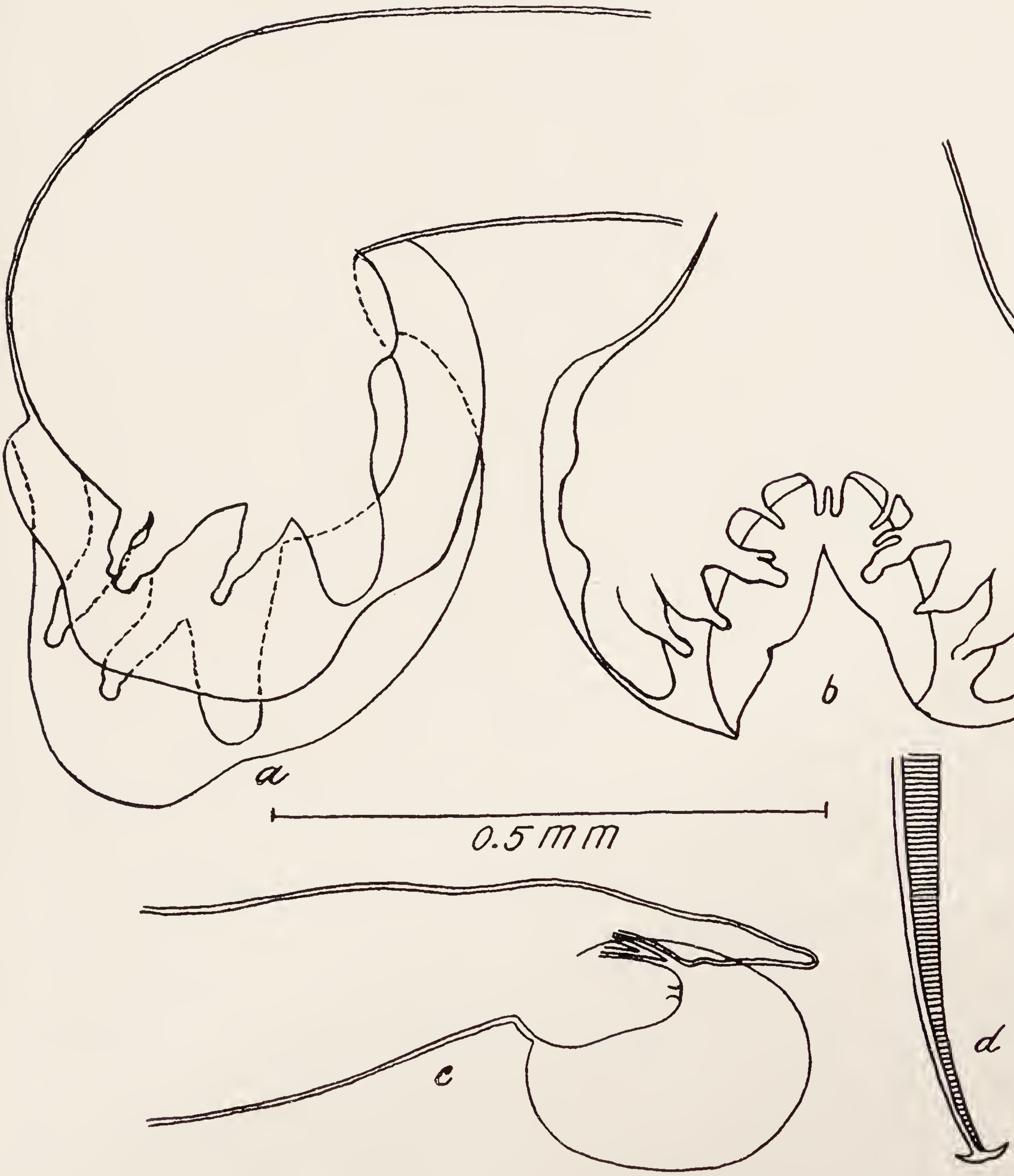


Fig. 77. *Choerostrongylus brevivaginatus*. *a*, male tail, lateral view; *b*, male tail, dorsal view; *c*, female tail, lateral view; *d*, distal end of spicule. From Gedoelst, 1923.

ARDUENNA STRONGYLINA (Rudolphi, 1819)

Railliet and Henry, 1911

The strongyline stomach worm of swine

Synonyms.—*Spiroptera strongylina* Rudolphi, 1819.

Hosts.—Primary: Swine, wild boar and cattle (reported once from cattle in the United States by Dikmans) and, by experimental feeding, guinea pig (developed to fourth stage larva; report of Ransom and Raffensperger); secondary: Coleoptera (beetles of the genera *Onthophagus* and *Aphodius*, including *O. hecate*, *A. castaneus* and *A. rufus*). Foster has pointed out that the worms first reported from the peccary as belonging in this species were subsequently found to be *Physocephalus sexalatus*.

Location.—Stomach, especially in the pyloric region, and small intestine.

Morphology.—*Arduenna*: White worms, filiform. Cuticle densely striated transversely. On the left side is a cuticular wing extending from a point about 280μ from the head to a point about 2 mm. from the tail end; this wing is about 35 μ in maximum width. Mouth about 45μ in diameter, with 2 lateral lips, each divided into 3 lobes and with a small papilla at the base of each lobe; just below the lips 2 chitinous teeth project into the mouth cavity. The pharynx is 83 to 98μ long by about 29μ wide, and is marked on the inside by a multiple spiral of chitinous ridges (Fig. 78). Esophagus 3.1 to 3.7 mm. long.

Male 1 to 1.5 cm. long by 300 to 387μ in maximum width just anterior to the caudal alae. Pharynx usually with 2 to 3 spirals. Caudal alae (Fig. 79) are about 1.2 mm. long, irregularly ovate, asymmetrical, the right wing about twice as wide as the left. There are 5 pairs of caudal papillae, asymmetrically arranged, of which 4 pairs are preanal and 1 pair postanal. Alae with fine longitudinal striae. Two unequal spicules, each grooved on the ventral surface; the left is 2.24 to 2.95 mm. long, very slender, ending in a fine point; the right is about 1/5 as long as the left, 457 to 619μ, and wider, about 10μ wide at its base. Cloacal aperture 155 to 200μ from the tip of the tail and surrounded on the left side and posteriorly by a cuticular thickening, serrate on its outer edge.

Female 1.6 to 2.2 cm. long by 368μ in maximum width one-third of the body length from the anterior end. Width gradually diminishes in the posterior third of the body, abruptly thinning a short distance in front of the anus to form a bluntly conical tail (Fig. 80). Pharynx usually with 4 to 5 spirals. Anus 215 to 275μ from the tip of the tail.

Vulva orbicular, slightly anterior to the middle of the body and near the lateral wing on the left side. Vagina 1.7 mm. long, extending posteriorly. Eggs oval, 34 to 39μ long by 20μ wide, thick-shelled, the shell surrounded by a thin irregular membrane, and contains a well developed embryo when deposited (Fig. 81).

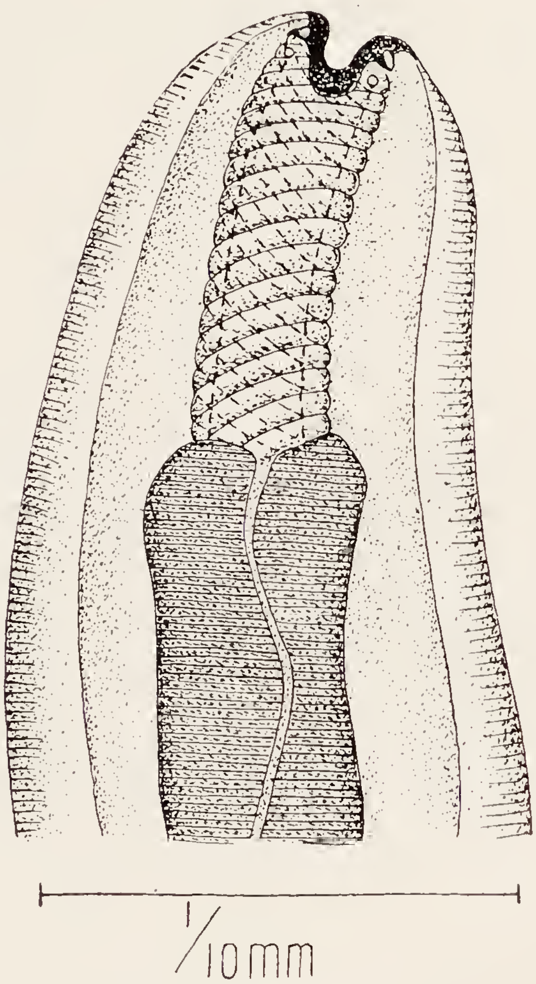


Fig. 78. *Arduenna strongylina.* Head end. Dorso-ventral view. From Foster, 1912.

Larva very similar to that of *Physocephalus sexalatus* (See page 109). The third stage larva is 1.9 to 2.8 mm. long by 80μ thick; tail 84μ long, terminating in a smooth knob not spined; esophagus 780μ long; cervical papillae placed very asymmetrically.

Life history.—The eggs are ingested by an insect, the Coleoptera (beetles) being the group involved, and the larvae develop to the infective stage (encapsuled third stage larvae) in the body cavity of the insect. These insects are then eaten by swine and the larval worms develop to maturity.

Distribution.—United States (common and widely distributed), South America (Argentina), Europe (Germany, France, Hungary, Roumania and Italy), Asia (Turkestan and Cochin-China), Africa (Algeria) and Australia.

Pathology.—Some writers have regarded these worms as comparatively harmless. Others have reported epizootics of a serious nature among swine as due to the injurious effects of these worms. The symptoms attributed to them are loss of appetite, terminating in complete anorexia, great eagerness for water, and restlessness. The

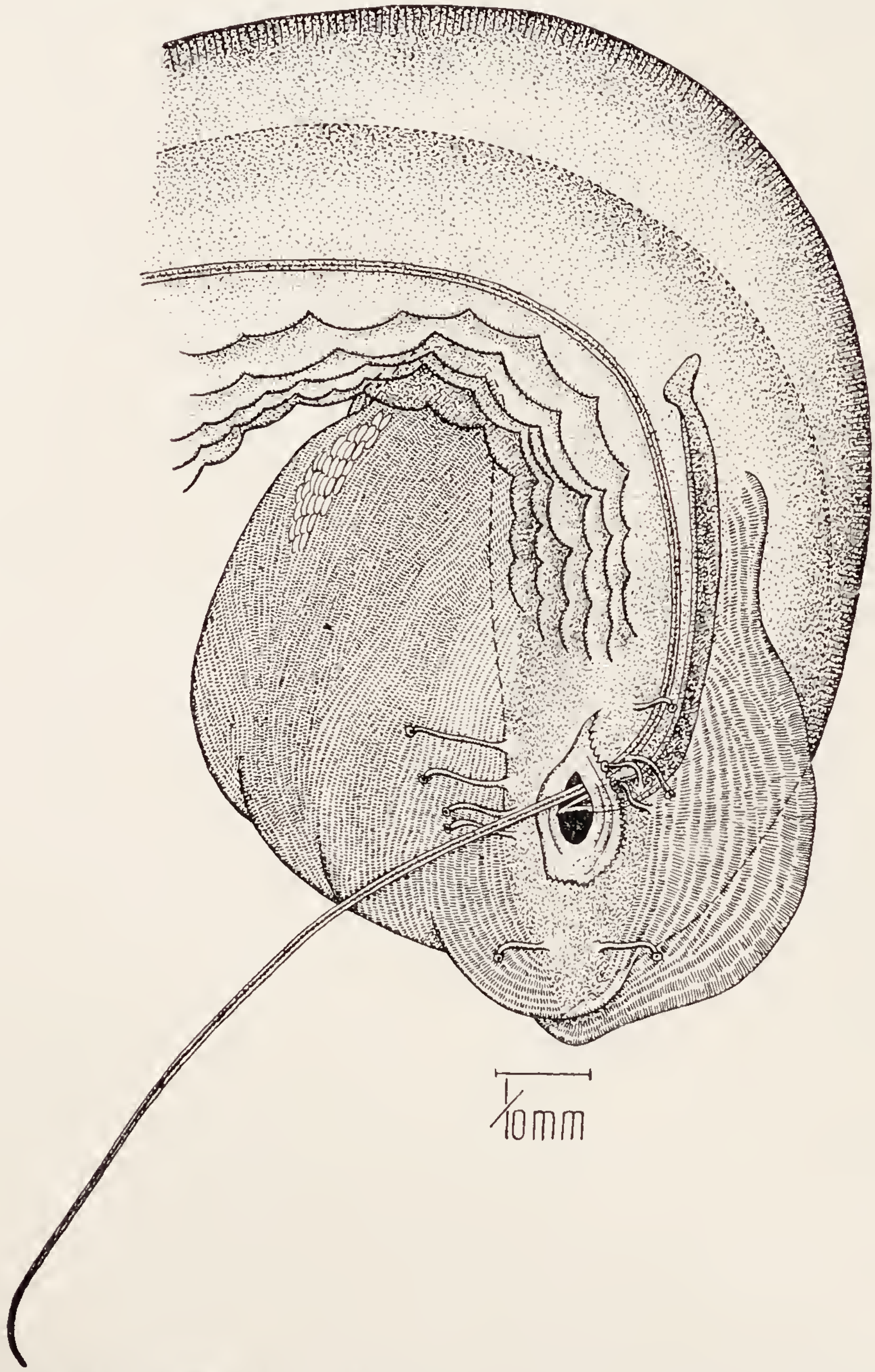


Fig. 79. *Arduenna strongylina.* Male tail. Ventral view. From Foster, 1912.

post-mortem lesions reported are: The formation of a thick, lamellar pseudo-membrane, firmly adherent to the pyloric stomach; under this is a superficial loss of mucous membrane; in this area numerous worms fastened partly in the stomach wall and partly in the pseudo-membrane; dark red areas, attaining the size of 2 to 3 cm. in diameter,

contain numerous worms, these areas being regarded by some pathologists as ulcers. The necrotic pseudo-membranes are regarded as possibly due to the action of the *Bacillus necrophorus,* which is enabled

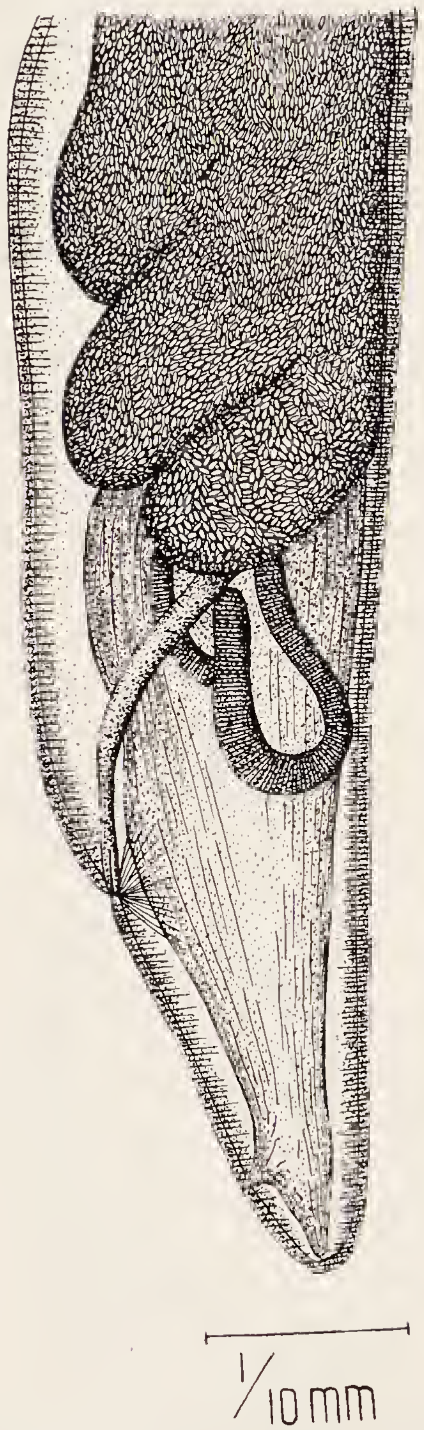


Fig. 80. *Arduenna strongylina.* Posterior end of female. Lateral view. From Foster, 1912.

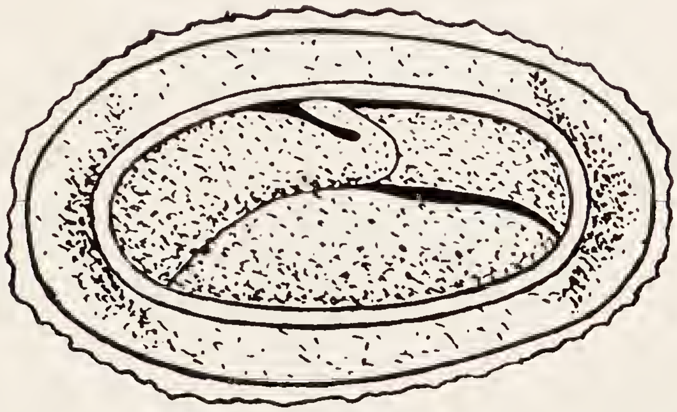


Fig. 81. *Arduenna strongylina.* Egg. x 1450. From Foster, 1912.

to enter and attack the mucosa where it has been wounded by the worms; the bacillus has been found, as would be expected, in these lesions. A worm 1.2 cm. long has been found by Foster to penetrate diagonally a distance of 1 cm. into the mucosa.

Treatment.—Youatt has recommended turpentine and salt in the food for removing these worms. Copper sulphate solution, in the dose recommended for sheep, 50 to 100 cc. (See Parasites of Sheep), or oil of chenopodium in the dose recommended for swine ascarids (See page 59) would perhaps be of some value in removing these worms. A high degree of efficacy is not to be anticipated, for the reason that the worms are evidently well protected for the most part by the pseudomembrane and are somewhat inaccessible to the drugs as a result of the habit of burrowing into the mucosa. Carbon tetrachlorid might be of some value.

Prophylaxis.—The sanitary measures recommended for the control of swine ascarids (See page 60), frequent and thorough cleaning out of the manure being a measure of prime importance. Other prophylactic measures must be based on control of the intermediate hosts.

ARDUENNA DENTATA (von Linstow, 1904)

Railliet and Henry, 1911

The toothed stomach worm of swine

Synonyms.—*Spiroptera dentata* von Linstow, 1904.

Hosts.—Primary: Swine, also *Sus cristatus;* secondary: Unknown, probably insects, especially Coleoptera (beetles).

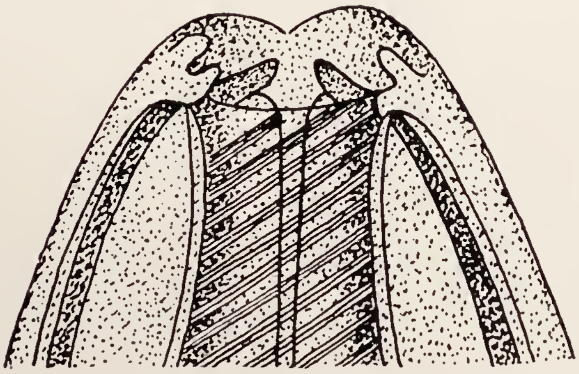


Fig. 82. *Arduenna dentata.* Head. Enlarged. From Foster, 1912, after von Linstow.

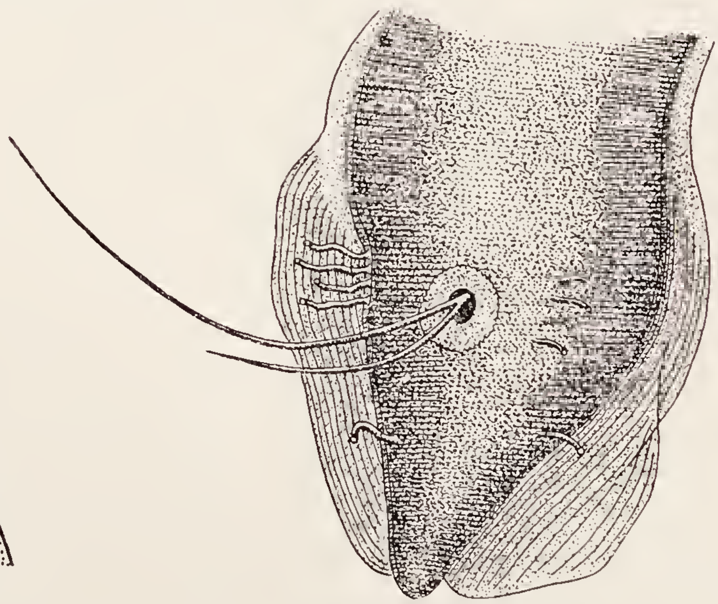


Fig. 83. *Arduenna dentata.* Male tail. Enlarged. From Foster, 1912, after von Linstow.

Location—Stomach.

Morphology.—*Arduenna.* Cuticle finely striated transversely. Mouth a transverse slit, with 2 trilobed lips, each lobe bearing a papilla. At

the base of the mouth and the entrance to the pharynx are a dorsal and a ventral tooth (Fig. 82). The pharynx is provided with spiral cuticular ridges.

Male 2.5 cm. long by 790μ in maximum width. The tail is coiled in a manner similar to that of *Ard. strongylina* (See page 102). Two unequal spicules (Fig. 83), the longer 920μ long, according to v. Linstow, or 3.75 to 4.23 mm., according to Railliet and Henry's measurements on specimens from swine; the shorter 350μ long, according to v. Linstow, or 540 to 650μ, according to Railliet and Henry, this spicule bearing a barb on its point. A group of 4 adanal papillae on each side and at some distance posterior to the cloacal aperture a pair of postanal papillae, all stalked. The cloacal aperture is surrounded by a broad chitinous ring, its external margin dentate. The caudal alae present longitudinal rows of scales.

Female 5.5 cm. long by 1.1 mm. in maximum width. The short conical tail is curved dorsally. The vulva is placed far posteriorly, dividing the body in the ratio of 70 to 23. Eggs small, thick shelled, and cylindrical, 39μ long by 17μ wide.

Life history.—Unknown; probably involves stages in an intermediate host, perhaps beetles, which eat the eggs and in which the infective larvae develop; these infected insects are probably swallowed by swine in feeding or drinking, the larval worms developing to adults in the stomach of the swine.

Distribution.—Annam, Cochin-China.

Pathology.—Unknown; perhaps similar to that for *Ard. strongylina* (See page 104). Brau and Bruyant found this worm in 2 per cent of the swine examined by them at Saigon.

Treatment.—Unknown; see remarks on *Ard. strongylina* (See page 106).

Prophylaxis.—Unknown; see remarks on *Ard. strongylina* (See page 106).

PHYSOCEPHALUS SEXALATUS (Molin, 1860) Diesing, 1861

The six-winged stomach worm of swine

Synonym.—*Spiroptera sexalata* Molin, 1860.

Hosts.—Primary: Swine (*Sus scrofa domestica*), also wild boar, white-lipped peccary and tapir. Dikmans has reported it from cattle in the United States. Seurat reports it from the ass, peccary and dromedary, and regards the ass as the normal host in Algeria. Foster notes

that Seurat's measurements do not agree with those for the worm from swine and questions the identification. Secondary hosts: Coleoptera (beetles), including *Scarabaeus* (*Ateuchus*) *sacer*, *Scar.* (*At.*) *variolosus*, *Gymnopleurus sturni*, *Geotrupes douei*, *Onthophagus nebulosus*, and *Ont. bedeli*; as encapsulated aberrant larvae, these worms occur frequently in various amphibia, reptiles, birds and mammals.

Location.—Stomach and small intestine of primary host; encapsulated in body cavity of secondary host; encapsulated in wall of intestine or in mesenteries in accidental hosts.

Morphology.—*Physocephalus*: Slender worms. Head about 60μ in diameter anteriorly, with 2 trilobed lips, each lobe bearing a thick, round papilla. Cuticle of head inflated for a distance of about 230μ from the anterior end. Cylindrical pharynx 263 to 215μ long by 53μ wide, provided with a single spiral ridge, which may break into separate rings in its middle portion and with from 21 to 25 turns to the spiral (Fig. 84). There are 3 lateral cuticular wings on each side, commencing at the base of the cephalic inflation and extending posteriorly about one-third of the body length. The middle wing on each side is about 60μ wide near the middle, and the other wings are about half as wide.

Male 6 to 9 mm. long by 315μ in maximum width where the lateral wings are widest. The narrow caudal membranes are about half the width of the body and are 1.4 to 1.5 mm. long. Caudal extremity twisted into a fairly regular spiral, usually with 3 turns. Long spicule (Fig. 84) 2.1 to 2.25 mm. long, grooved on its ventral surface, very slender, tapering to a very fine point; short spicule 300 to 350μ long, relatively broad at its base and tapering suddenly to a fine point, with its ventral surface provided with a narrow wing extending almost to the tip. Caudal alae with 4 pairs of long-stalked preanal papillae and with 4 pairs of very small, short-stalked postanal papillae in a group close to the tip of the tail.

Female 1.3 to 1.9 cm. long with a maximum width of 333 to 450μ near the anus. The body increases in width in the region of the lateral wings, then diminishes rapidly to half as much, then increases to the maximum at the anus, then abruptly diminishes, the body ending in a blunt point with a mucronate tip (Fig. 86). Anus 120μ from the tip of the tail. Vulva posterior to the middle of the body, dividing the body in the ratio of 9 to 8. Vagina extends posteriorly. Two uteri, the ovaries at opposite extremities. Eggs, elliptical, 34 to 39μ long by 15 to 17μ wide, slightly flattened at the poles, and containing a well

developed embryo when deposited; the shell is surrounded by a thin irregular membrane. It is very similar to that of *Arduenna strongylina* (Fig. 81).

Embryo.—130μ long, with an obtuse anterior extremity provided with a perforating lancet, and with a pointed tail.

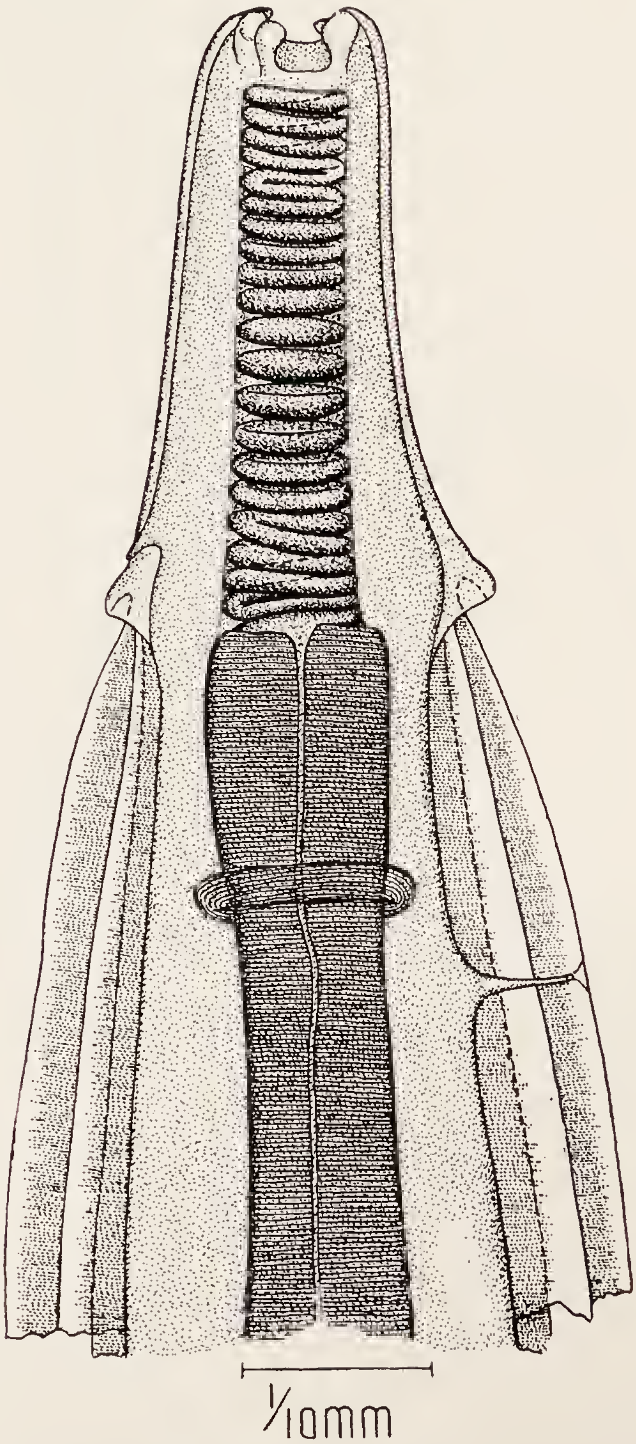


Fig. 84. *Physocephalus sexalatus.* Anterior end of body. Dorsal view. From Foster, 1912.

Larva. The *first stage larva* is the embryo from the time of hatching until its first molt. This larva makes its way through the wall of the intestine of the intermediate host and gets to the body cavity. Here

it becomes larger, 420μ long by 40μ wide; the tail is 60μ long, bluntly conical and terminates in a small mucron; the worm presents a brilliant, piriform excretory vesicle towards its anterior fourth. This worm molts

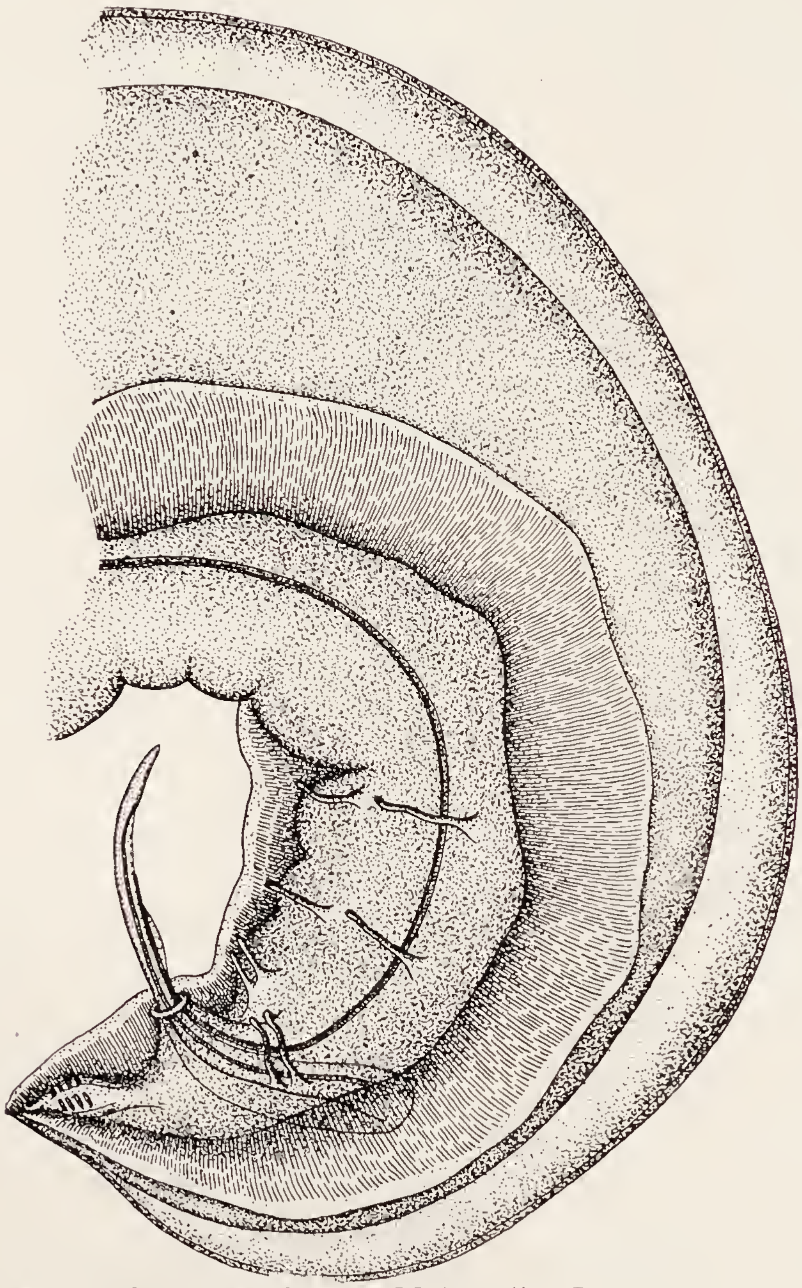
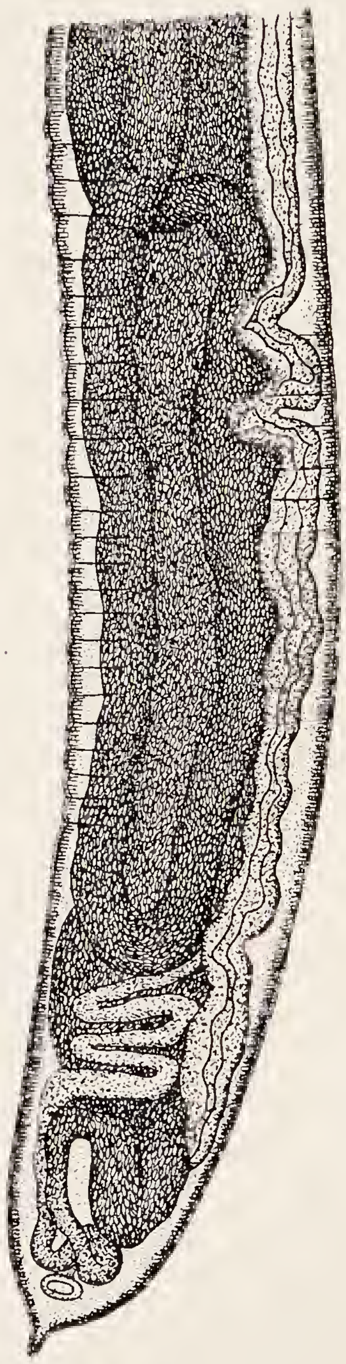


Fig. 85. *Physocephalus sexalatus.* Male tail. Latero-ventral view. x 100. From Foster, 1912.

to form the *second stage larva* (Fig. 87). This larva attains a length of 870μ and is 42μ wide. It is at first free and encapsulates just before the next molt. The head is rounded and without a lancet; the tail is rounded and without a mucron; the anus is on a distinct elevation; the esophagus is a third of the body length; the genital primordium is on the ventral surface of the intestine near the middle of the body. When the new cuticle is formed, the larva encapsulates, the capsule forming by an inflammatory reaction of the trachea of the host insect; capsules

contain usually 1 larva, but may contain up to 7. In the capsule the larva develops to a *third stage larva.* This larva is coiled in a spiral or a figure of 8, and has its shed cuticle alongside of it; it does not differ materially from the preceding form, but attains a length of 940μ to



1/10 mm

Fig. 86. *Physocephalus sexalatus.* Posterior end of female. Ventral view. From Foster, 1912.

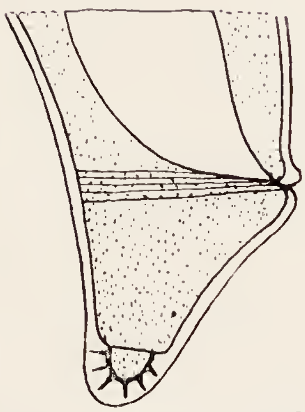


Fig. 87. *Physocephalus sexalatus.* Tail of second stage larva showing enclosed tail of third stage larva. Enlarged. From Seurat, 1916.

1.81 mm. The tail ends in a knob studded with spines. When this larva is swallowed by an animal other than the normal host (See aberrant hosts, page 108), it does not undergo further development, but

encapsulates in the wall of the intestine or in the mesenteries. When swallowed by an animal capable of functioning as a primary host, it molts and forms a *fourth stage larva.* This larva is from 2.5 long to 5.0 mm. long for males and 9 mm. long for females; it shows the pharyngeal ridges of the adult and, in a late phase of this stage, the 2 lateral alae. On undergoing a fourth molt, it becomes adult.

Life history.—The eggs passed in the feces of the primary host do not hatch until swallowed by the intermediate host. In this host, the larvae develop as noted in the above descriptions to encapsuled third stage larvae in the body cavity. When beetles are swallowed, usually in the act of coprophagy on the part of the primary host (Seurat notes coprophagy on the part of the poorly nourished donkey in Algeria), the larvae molt twice and become mature worms. In accidental hosts, the third stage larvae again encyst in the walls of the intestine or in the mesenteries. Seurat has counted 4,880 larvae of this worm in 1 beetle, *Scarabaeus* (*Ateuchus*) *sacer,* the same beetle harboring 68 larvae of *Spirocerca sanguinolenta.*

Distribution.—United States, Brazil, Italy, Germany, Roumania, Algeria, Madagascar, Indo-China and Australia.

Pathology.—So far as is known at present, the pathological conditions due to this worm are apparently very similar to those due to *Arduenna strongylina* (See page 104).

Treatment.—See remarks on *Arduenna strongylina,* page 106).

Prophylaxis.—This is based on sanitation, especially on measures to prevent coprophagy. In view of the fact that the various beetles known to function as intermediate hosts are dung beetles, spending a large part of their lives in dung, the eating of manure by swine or other animals must be prevented by suitable disposal of the manure. Frequent and thorough cleaning of pens and yards is a measure of value. Where this cannot be done frequently, measures to hasten the drying of the manure will render it less suitable to beetles for food or breeding purposes; dragging tree branches through manure is one method of hastening drying. The process of following corn-fed cattle with swine to clean up undigested grain is a measure that favors the spread and intensity of infection with this worm.

SIMONDSIA PARADOXA Cobbold, 1864

Simondsia

Hosts.—Primary: Swine; secondary: Unknown, probably arthropods, possibly beetles.

Location.—Stomach; males free in lumen according to Cobbold, and in mucosa with head and tail out, according to Colucci and Arnone; females in small tumors or cysts in the stomach wall with head projecting into the lumen through an aperture, the mucosa moulded about the dilated posterior part of the worm.

Morphology.—*Simondsia*: Sexes dimorphic. Head with 2 strong lips and 2 papillae. Two cervical alae present. Pharynx with a chitinous spiral ridging (Fig. 88).

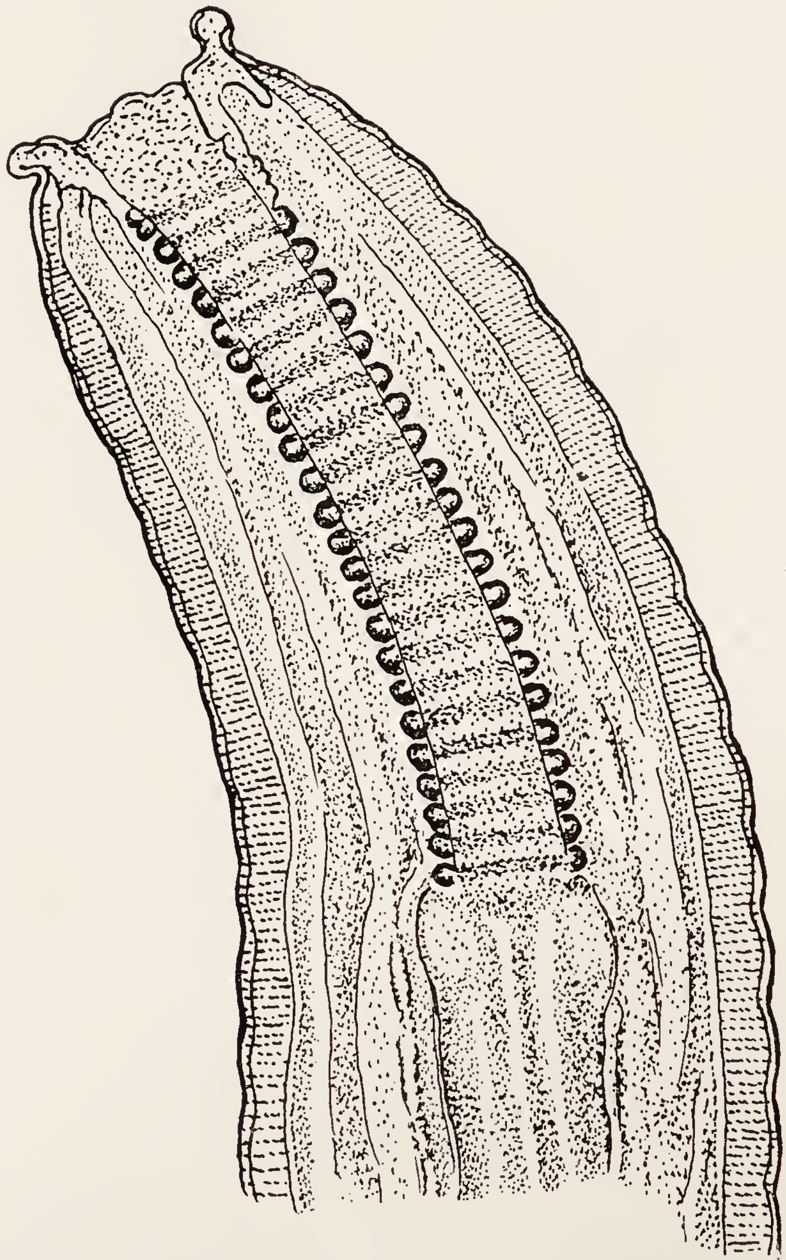


Fig. 88. *Simondsia paradoxa.* Anterior end of body. x 360. From Colucci and Arnone, 1896.

Male 1.2 to 1.5 cm. long, very slender, and with the tail end wound in a spiral (Fig. 89). Spicules unequal, long and slender, according to Cobbold; Colucci and Arnone state that there is 1 spicule (Fig. 90) transversely striate and with a truncated termination.

Female 1.5 mm. long, the body becoming wider posteriorly, and bearing a large rosette-shaped structure (Fig. 91) situated anterior to the tail and containing the much dilated intestine and parts of the 2 genital tubes (Fig. 92). The tail follows this rosette and is twice as thick as the anterior portion of the body; this is a massive muscular structure, the rectum surrounded by a wide spiral. Directly in front of the anus are 3 spines with broad bases. The vulva is a transverse

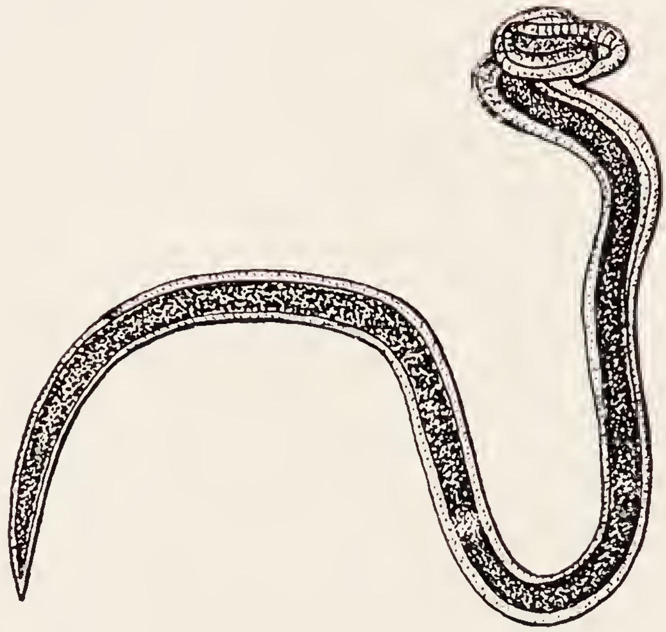


Fig. 89. *Simondsia paradoxa.* Male. Entire worm. Enlarged. From Cobbold, 1883.

slit, with a prominent posterior lip, and is in the anterior third of the body. Eggs (Fig. 93) ovoid or ellipsoidal, often contracted at the middle, and 28 to 29μ long.

Life history.—Unknown; probably involves intermediate stages in some arthropod, which swallows the eggs of the worm in feces of infested swine and becomes infested with larvae which develop to a stage infective for swine when the infested arthropods are swallowed by swine. Colucci and Arnone found immature worms 650μ long by 20 to 25μ wide, with dilated intestine in an enlarged posterior part of the body, in hog wallows and think these may be larvae of this worm.

Distribution.—England, Hungary and Italy.

Pathology.—These worms form small tumors or cysts 2 to 3 mm. high, in the stomach wall, the male in a burrow in the mucosa and the female in an elevated fibrous cyst, involving the mucosa and muscularis, with a thickening of the serosa, accompanied by local endarteritis and thrombosis. Where the worms are numerous, it may be assumed with considerable likelihood that they would cause a chronic gastritis, with catarrhal secretions due to the abnormal functioning of the persistently

irritated mucosa. Joest states that a chronic catarrhal condition is present. It would naturally follow that the functions of digestion would be interfered with as regards secretion and absorption, and this in turn

Fig. 90. *Simondsia paradoxa.* Posterior end of male. Lateral view. x 105. From Colucci and Arnone, 1896.

would have its sequel in malnutrition. With large numbers of worms, especially in young pigs, the effects might be quite severe. However, it has been reported from animals that showed no evident symptoms

and no evidence of gastritis. In Italy infested swine were found not well nourished and there was a coincident skin disease.

Treatment.—Unknown. To the extent that most of the female worm is protected by its position in the tumor, the removal of these

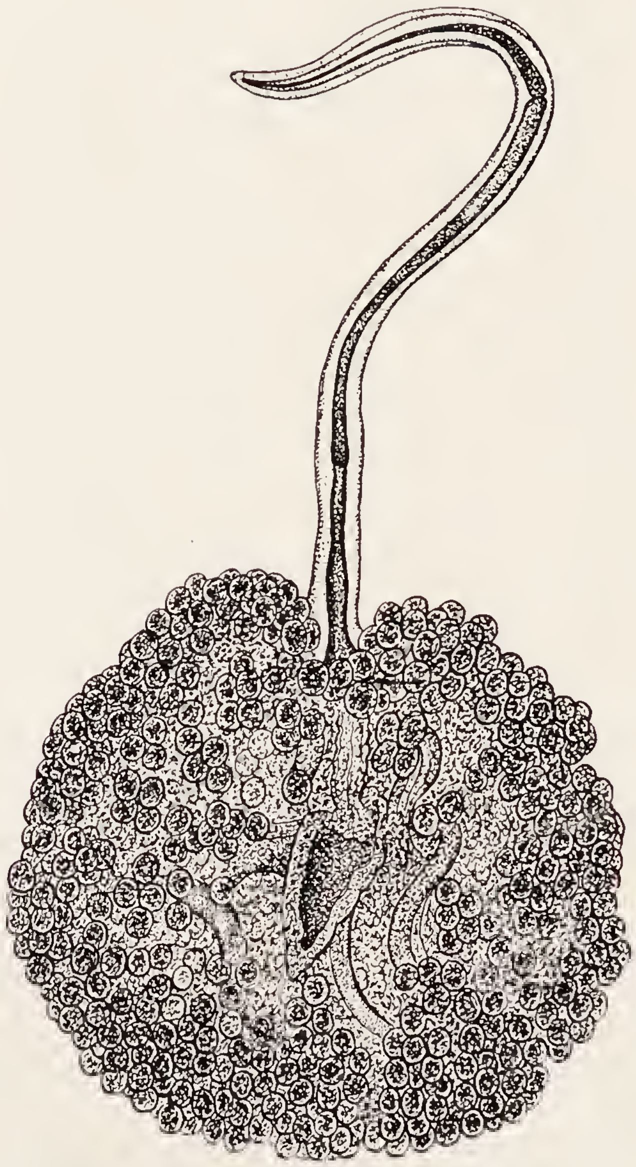


Fig. 91. *Simondsia paradoxa*. Female. Entire worm. Enlarged. From Cobbold, 1883.

worms may prove very difficult. However, it is likely that the male worm, and possibly the females to some extent, would prove amenable to treatment with 1-dram doses of oil of chenopodium.

Prophylaxis.—Unknown. Sanitary measures, especially those looking toward prompt and frequent removal and proper disposal of manure, as bearing the infective agents in the form of eggs, are indicated. Control of coprophagous insects, as noted under prophylaxis for *Physocephalus sexalatus* (See page 112) is also indicated.

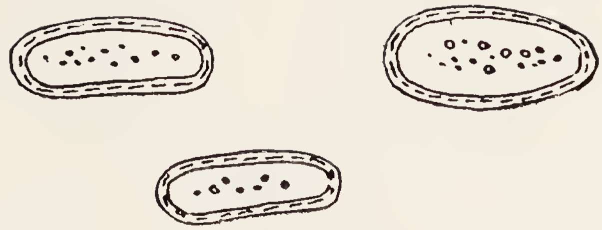


Fig. 93. *Simondsia paradoxa.* Eggs from uterus. x 500. From Railliet, 1893, after Cobbold.

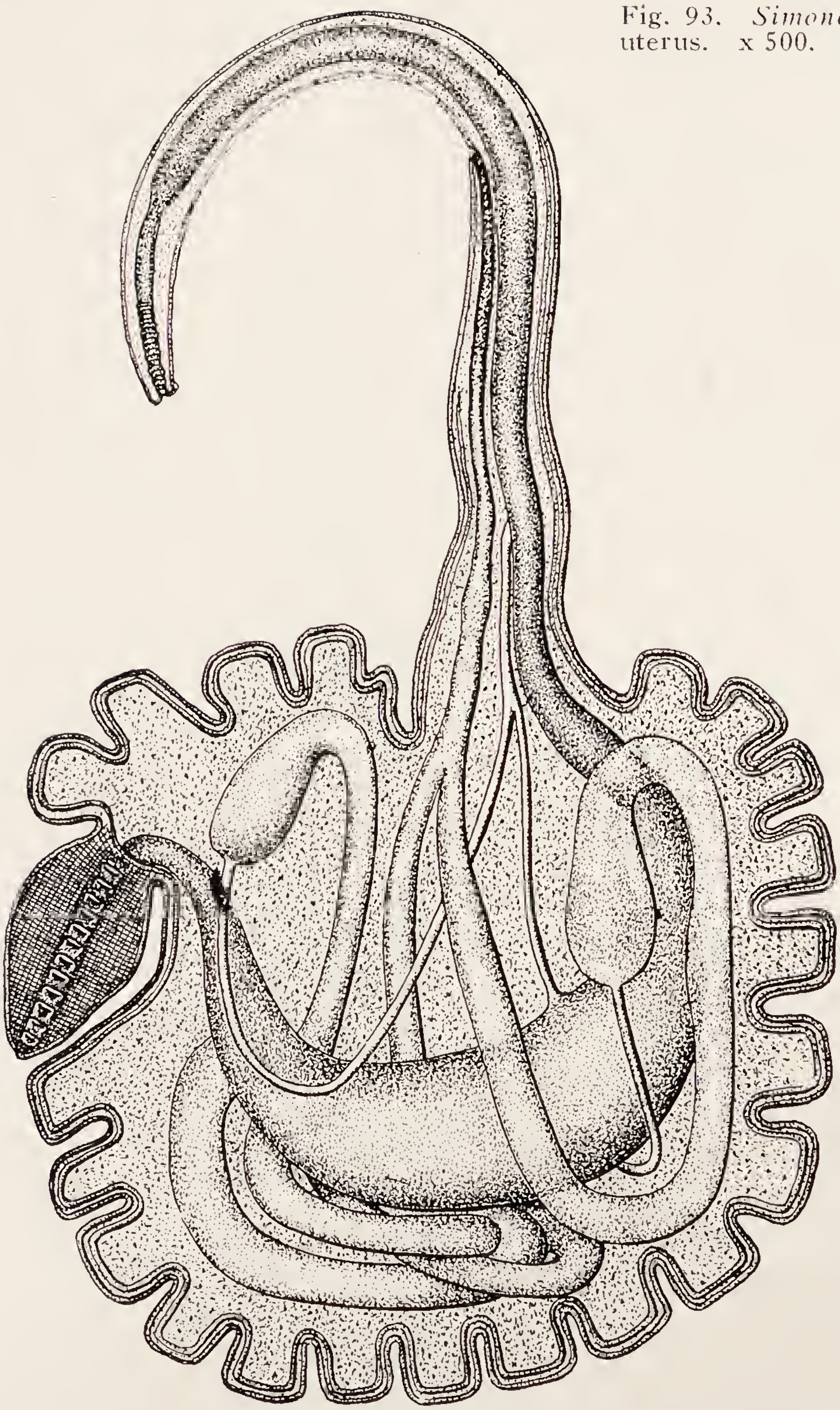


Fig. 92. *Simondsia paradoxa.* Female. Entire worm, showing internal structures. x 20. From Colucci and Arnone, 1896.

GONGYLONEMA PULCHRUM Molin, 1857

The gullet worm of swine

Synonyms.—This worm is regarded by some writers as identical with *G. scutatum*, and not a distinct and valid species. Seurat regards *G. confusum* Sonsino, 1896, from the horse as a synonym of *G. pulchrum*. Note that this latter is also regarded as *G. scutatum*, which occurs in the horse. See the related species, *G. ransomi*.

Hosts.—Primary: Swine, also wild boar, horse, and, possibly, man; secondary: Probably coprophagous beetles. A *Gongylonema*, regarded as possibly this species, was reported from the buccal mucosa

Fig. 94. *Gongylonema pulchrum*. Entire worm. Natural size. From Molin, 1857.

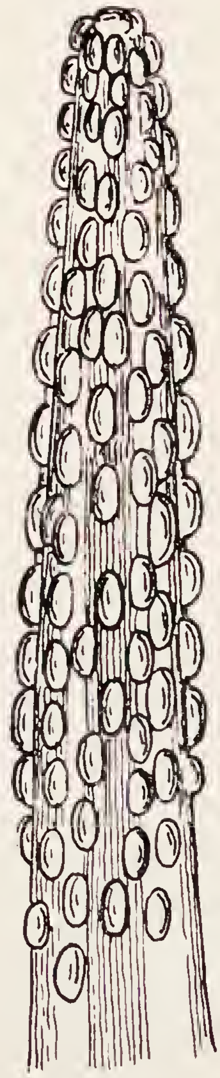


Fig. 95. *Gongylonema pulchrum*. Anterior end of body. Enlarged. From Molin, 1857.

of man in the United States by Ward; 2 similar cases have since been reported by Stiles and 1 by Ransom. Owing to the possible specific distinctness of this form, Stiles has provisionally named the form from

man *G. hominis*. If this is identical with the form in American swine it is *G. ransomi*, a name antedated by *G. hominis*.

Location.—In the mucosa of esophagus and pharynx in primary host; encapsulated in the abdominal cavity of the secondary host.

Morphology.—*Gongylonema*: Long slender worms (Fig. 94), very similar to *G. ransomi* (See page 120). Neumann notes that it differs from *G. scutatum* in its constantly smaller dimensions. Cuticular tuber-

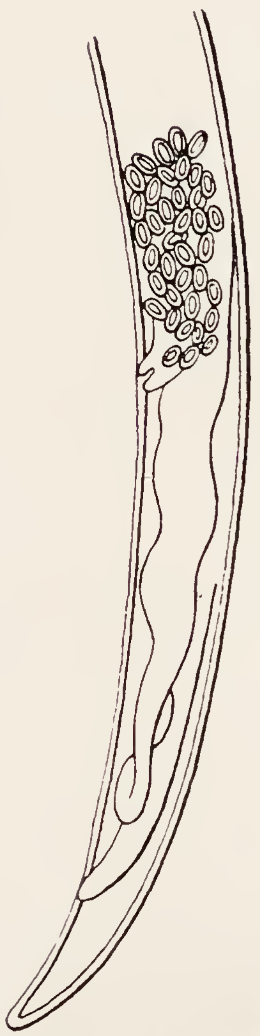


Fig. 96. *Gongylonema pulchrum*. Posterior end of female. Lateral view. Enlarged. From Molin, 1857.

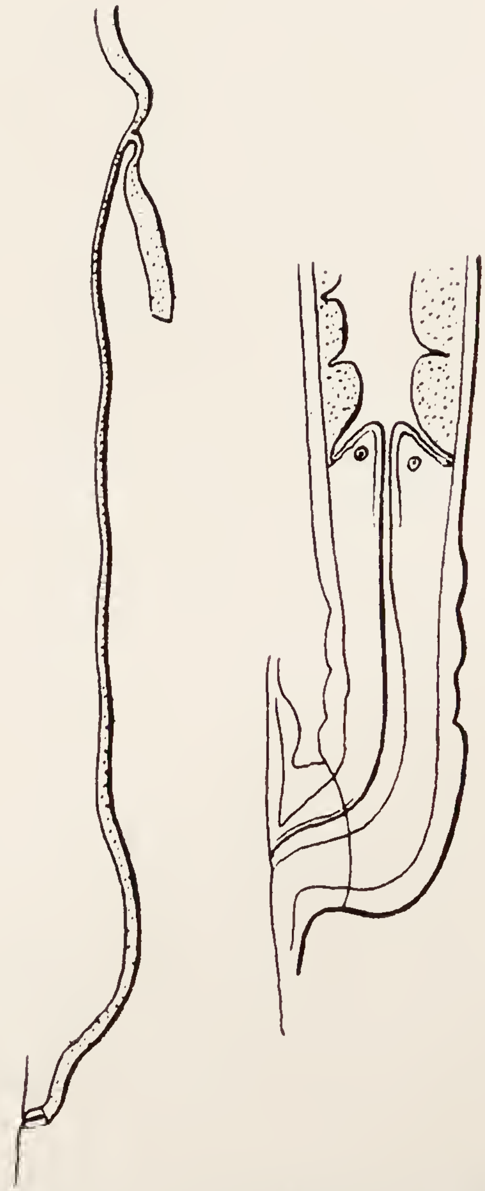


Fig. 97. *Gongylonema pulchrum*. Left, ovejector and uterus; right, vestibule and sphincter. Enlarged. From Seurat, 1912.

cles (Fig. 95) over cephalic and cervical area 700μ long, 2 rows in each of 4 submedian fields. Excretory pore opens on transverse plaque. Lateral alae extend from cervical papillae to just posterior to area of cuticular tubercles.

Male 1.4 to 1.925 cm. long by 175 to 195μ wide. Left spicule 4 to 5 mm. long; right spicule 84 to 110μ long.

Female 3.7 to 4 mm. long by 350μ wide. Vulva about 2 mm. from the tip of the tail (Fig. 96). The vestibule and sphincter of the vagina (Fig. 97) have a total length of only 200μ and are not delimited from one another; the trompe, or third portion of the vagina-ovejector is 13 mm. long, of uniform diameter, and extends anteriorly to meet the divergent uteri. Eggs 52 to 56μ long by 32μ wide.

Life history.—The eggs pass in the feces of the primary host and are probably swallowed by coprophagous beetles as they feed on the manure. Very likely the same species that serve as secondary hosts of *G. ransomi* (See page 121) will serve as secondary hosts fo the gullet worm of swine also. The probable life history is as follows: In the hosts mentioned, or in croton bugs fed on these eggs, the eggs probably hatch and the first stage larva makes its way to the body cavity, where it molts twice to form a third stage larva, which is encysted. When these infected beetles are swallowed by swine, the larval worms become adults and occur sewn in serpentine burrows in the esophageal and pharyngeal mucosa.

Distribution.—Europe and Algeria. The gullet worm commonly found in swine in the United States is not this species, but is *G. ransomi.*

Pathology, treatment, etc.—Similar to those for *G. ransomi* (See page 121).

GONGYLONEMA RANSOMI Chapin, 1922

The American gullet worm of swine

Hosts.—Primary: Swine; secondary: Croton bugs (*Ectobia germanica*), in experimental feedings by Ransom and Hall, but probably coprophagous beetles, such as species of *Aphodius* and *Onthophagus*, in nature.

Location.—In burrows in the mucosa of the tongue and esophagus of primary host; in body cavity of secondary host.

Morphology.—*Gongylonema*: Cuticular tubercules (Fig. 98) round or oval to almost rectangular, arranged as follows: On the dorsal surface, 4 rows more or less evenly spaced but somewhat concentrated near the dorsal line; on the ventral surface, 4 complete rows crowded toward the median line, with an incomplete row on either side between the external and the lateral alae. No dorsal wing present. Lateral alae short, extending a short distance beyond the posterior limit of the

tubercles, slightly unequal in length. The cervical papillae are on circular tubercles in crater-like depressions just anterior to the cephalic end of the lateral alae. Excretory pore opens between the tubercles of the median ventral rows just anterior to the union of the muscular and non-muscular portions of the esophagus, not on a transverse scute as in other species.

Male averages 2.3 cm. long by about 150μ wide at union of esophagus and intestine. The cuticular tubercles present in the anterior 600 to 900μ of body length. Caudal alae (Fig. 99) enclose tip of tail; the right wing is somewhat longer than the left. A variable number of caudal papillae; usually 6 preanal and 4 postanal. Cloacal aperture 250μ from tip of tail. Caudal glands subterminal. Right spicule (Fig. 100) short and stout, slightly curved and twisted, 120μ long, measured between extremities in a straight line. Left spicule about 1 cm. long and very thin, infundibuliform at cephalic end, the length of this spicule separating this species from *G. pulchrum*. Gorgeret (or telamon) with 2 parts, a straight part lying parallel to the short spicule and about 55μ long, and an oval part lying dorsal to the spicules directly back of the cloacal aperture and 29μ long.

Female averages 3.7 cm. long by about 190μ wide at union of esophagus and intestine. Cuticular tubercules present in the anterior 1 to 1.3 mm. of body. Tail rather blunt and slightly curved. Caudal glands subterminal. Anus 200μ from tip of tail. Vulva slightly salient, 1.75 mm. from tip of tail. Vagina long, extending forward from vulva to near equator of body. Uteri divergent, extending nearly to extremities of worm. Eggs ovoid, 55.5μ long by 32μ wide, containing embryos when oviposited.

Life history.—Probably involves intermediate stages in coprophagous beetles. Ransom and Hall report feeding eggs from swine gullet worms to croton bugs, the infective larvae being found encysted at the end of a month in this host. As the common gullet worm of swine in the United States, and the only one definitely known to be present at this time, is *G. ransomi*, it seems safe to assume that they were dealing with this species.

Distribution.—United States. This worm is very common in the South, moderately common in the middle Atlantic States, middle West and Pacific Coast States and least common in the Rocky Mountain States and States along the Canadian border.

Pathology.—Ordinarily worms of this genus are only of interest as they concern the use of weazands for sausage casings. This species, however, commonly occurs in the tongues of swine and its occurrence

calls for measures to detect and remove it. The detection of the worm is facilitated by the use of a device first suggested and made by R. E. Learn and E. W. Murphy and consisting of a wooden skewer which

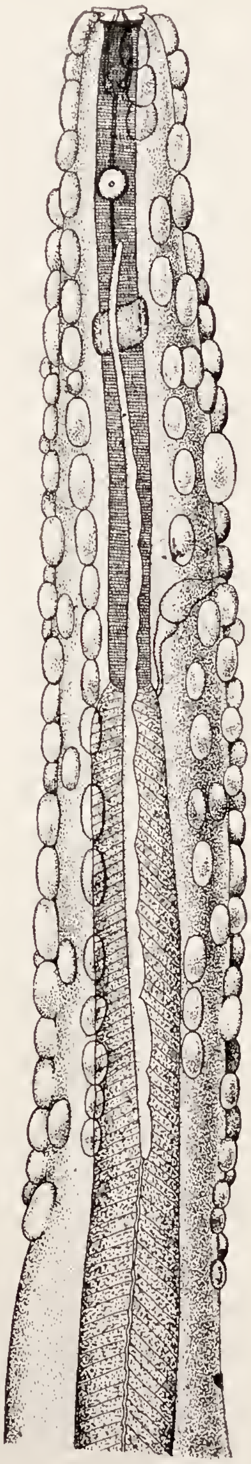


Fig. 98. *Gongylonema ransomi.* Anterior end of female. Enlarged. From Chapin, 1922.

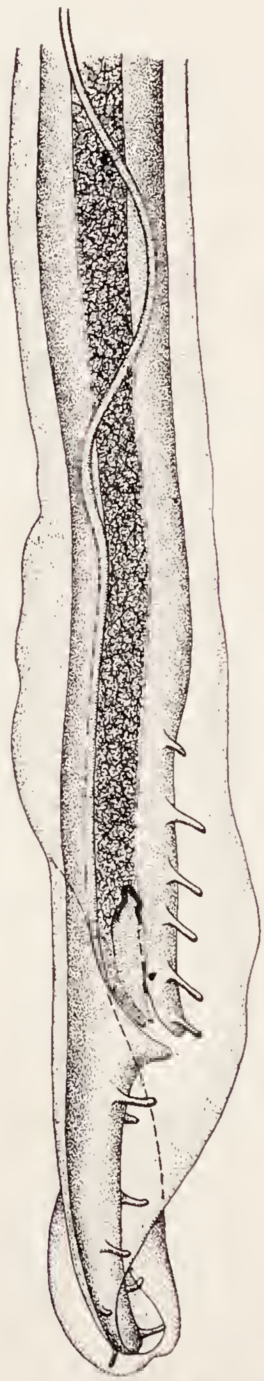


Fig. 99. *Gongylonema ransomi.* Posterior end of male. Enlarged. From Chapin, 1922.

Fig. 100. *Gongylonema ransomi.* Spicules and gorgeret. Enlarged. From Chapin, 1922.

has the end trimmed down to a short conical point and a pin driven into it, the head of the pin being then removed, the pin shortened to a little less than a quarter of an inch of projecting metal which is bent slightly

near the tip and ground flat on two sides. This is used to scratch shallow furrows in the mucosa of the dorsal surface of the tongue between the papillae at the root of the tongue and a line drawn across the tongue 2 or 3 inches in front of the vallate papillae. This instrument catches and pulls up the worms.

To remove the infested mucosa from the tongues they are scalded with water at a temperature of 145 degrees F. or higher and then drenched with cold water in a washer provided with baffles, similar to that used for scalding hog stomachs.

Treatment.—No treatment is yet known for removing these worms from animals.

Prophylaxis.—The indicated measures consist in preventing swine eating coprophagus beetles, a difficult and apparently impracticable procedure, and the provision of sanitary surroundings to prevent the exposure of swine to areas heavily contaminated with their manure and hence likely to have on it numerous infested insects. This is also a difficult matter, but the sanitary system for the control of ascarids in swine (page 60) is probably of some value in controlling infestations with gullet worms.

GNATHOSTOMA HISPIDUM Fedtschenko, 1873

The thorny gnathostome, or swine gnathostome

Synonyms.—*Cheiracanthus hispidus* (Fedtschenko, 1873) Csokor, 1882.

Hosts.—Swine, as adults and larvae; larval stages in birds (aberrant).

Location.—Stomach, free or attached to the gastric mucosa, especially in the fundus, as adults in swine; in liver and blood vessels as larvae in swine; encapsulated in subcutaneous connective tissue in birds.

Morphology.—*Gnathostoma*: Thick, cylindrical worms (Fig. 101); the body usually curved, and entirely covered with spines; on the head (Fig. 102) these spines are hooks arranged in 9 to 11 concentric circles with the points of the spines directed posteriorly; from the neck back to the middle of the body the spines are widened and each is divided posteriorly, according to Ciurea, into 3, 4, 9 or 11 points; Baylis and Lane say with 7 points; these points are subequal in anterior body region, the median point becoming the longest in the posterior region and the other points disappearing; the spines become smaller posteriorly and have longer and more slender points; behind the middle of the body are only bristle-like processes, the body being covered as if with fine hairs. In Austria the worm is called the tri-colored worm, the anterior

end being rose-red as a result of ingested blood, and the remainder of the body dark-grey from the pigmented intestine, streaked with yellow by the yellow genital tubes. The spherical head is followed by a deep cleft, the head being said to have a superficial resemblance to the head

Fig. 101. *Gnathostoma hispidum.* Left, female; right, male. Natural size. From Ciurea, 1911.

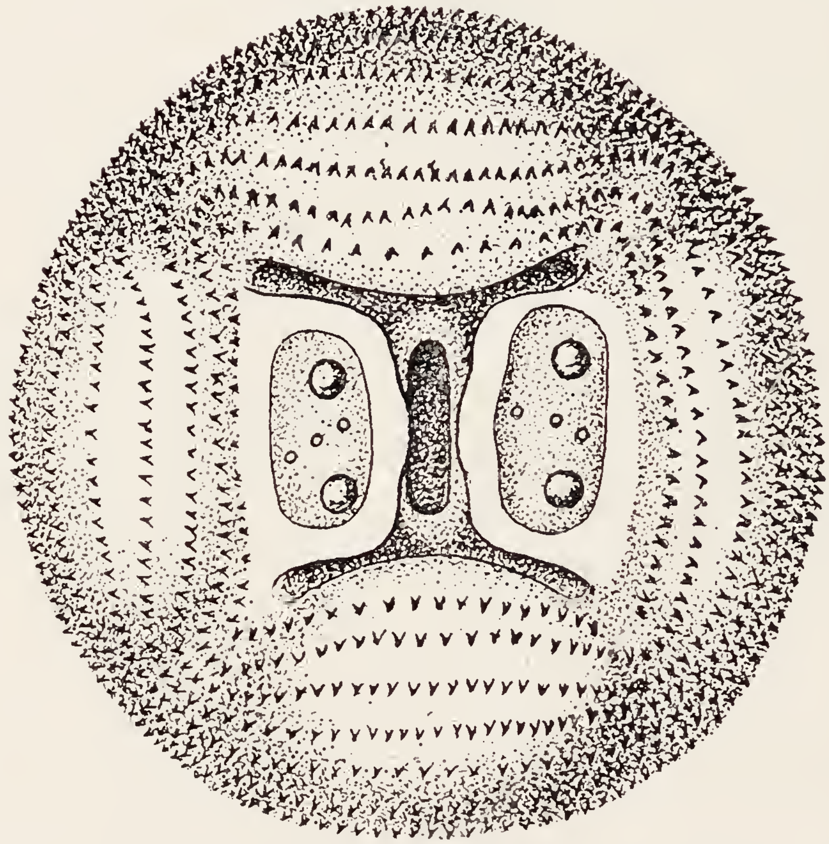


Fig. 102. *Gnathostoma hispidum.* Head. View from front. x 128. From Ciurea, 1911.

of a fly. The body is swollen just behind the neck and then diminishes to a cylindrical shape. From a head end view, the head shows a rectangular, almost quadratic depressed area in the middle. In the middle of this area is the elliptical mouth, with its long axis dorso-ventral; on each side of the mouth is a broad projecting lip, which bears 2 large papillae and a row of 3 small papillae between the large ones and crossing their axis rather asymmetrically.

Male 1.5 to 2.5 cm. long. The bursa (Fig. 103) is shaped like the bowl of a spoon and provided with 19 papillae, which Ciurea divides into genital papillae and sensory papillae. Those he regards as genital are stalked and piriform; of these there are 3 large ones on each side of the bursa, the middle one being in the mid-dorsal line, and 6 small ones, of which 2 are close together in front of the anus, 1 between the group of large ones and the middle-sized ones on each side, and 1 between the middle and the posterior members of the group of large ones on each side. Those he regards as sensory are sessile, 2 in front and 2 behind

the anus. Baylis and Lane restrict the genital papillae to 4 pairs of large lateral papillae and 2 pairs of small ventral papillae. The cloacal aperture is a transverse cleft, 312μ from the tip of the bursa. The 2 spicules are dissimilar, the left one slender and curved, 900μ long, the right one thicker and 400μ long. Baylis and Lane state that the left spicule is twice as long as the right spicule. Anterior of the cloacal aperture are 2 large, elliptical swellings which bear the 2 preanal sensory papillae and partly overlie the 2 preanal genital papillae. (The

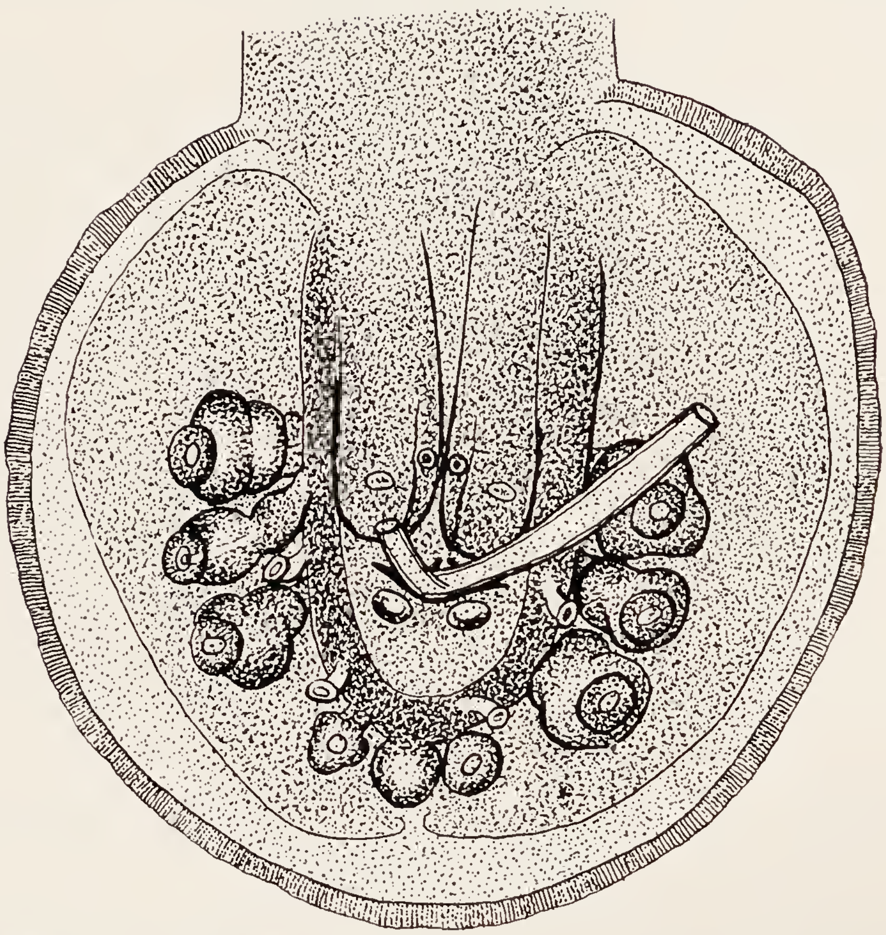


Fig. 103. *Gnathostoma hispidum.* Male tail. Ventral view. x 60. From Ciurea, 1911.

distinction between the 2 groups of papillae is open to question. All papillae are sensory and all those of the bursal region are presumably entitled to the designation of genital on the basis of morphology and function, whether stalked or sessile.)

Female 3.2 to 4.5 cm. long by 2.5 mm. wide in the dilated region behind the neck. The head is 800μ long and 1.73 mm. wide. The tail is bluntly conical. The vulva is slightly anterior to the middle of the body and has slightly salient lips, according to Ciurea; it is in the middle, according to Fedtschenko; and is slightly posterior, according to von Linstow; Baylis and Lane regard it as posterior. The eggs (Fig. 104) are 70 to 74μ long by 39 to 42μ wide, the shell marked with

small depressions, according to Ciurea, smooth, according to von Linstow, and with a wart-like projection or plug at one pole; segmentation begins in the anterior portion of the uterus.

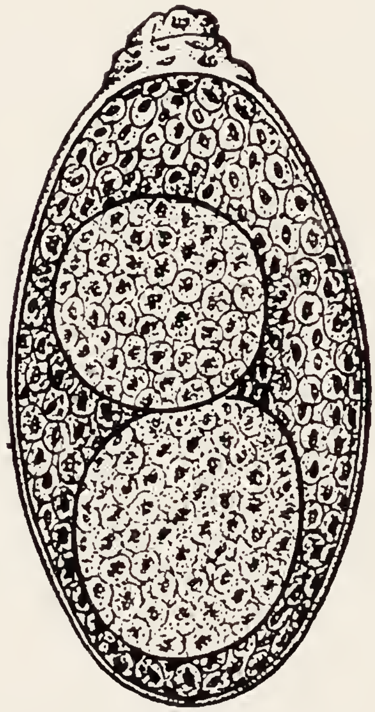


Fig. 104. *Gnathostoma hispidum.* Egg. x 700. From Ciurea, 1911.

Life history.—Unknown.

Distribution.—Europe (Hungary, Austria, Germany and Roumania) and Asia (Turkestan).

Pathology.—These worms bore deep into the mucosa, producing a round, sharply defined cavity the size of a pin head, surrounded by a reddened area, the base of the cavity containing blood or serum. The condition is one of localized inflammation and hemorrhage. Where the worms are numerous, infested animals may become cachectic, the gastric mucosa is thickened and uneven and the worms give rise to a chronic gastritis, with the accompanying lesions and symptoms.

Trattner has reported the larvae from the liver and its blood vessels (veins) in swine in 3 cases in Hungary. These larvae give rise to a chronic interstitial hepatitis. The interlobular connective tissue and the capsule of Glisson are thickened and heavily infiltrated with eosinophiles, these latter being also numerous about the blood vessels.

Treatment.—Unknown. It is likely that oil of chenopodium in the doses used for removing ascarids from swine (See page 59) would be effective in removing this worm. Copper sulphate, 1 per cent solution in water, in the dose recommended for stomach worms in sheep, 50

to 100 cc., (See Parasites of Sheep) might also be effective, provided its emetic action did not operate in swine to prevent the drug from exerting its effect on the worms.

Prophylaxis.—Unknown. Such general sanitary measures as are recommended for control of ascarids in swine (See page 60) will doubtless be of some value.

FILARIA BAUCHEI Railliet and Henry, 1911

Bauche's filarid

Synonym.—*Filaria hellemansi* Smit, 1920.

Hosts.—Primary: Swine; secondary: Unknown, probably some biting arthropod.

Location.—Lungs; pulmonary arteries.

Morphology—*Filaria*:

Male unknown.

Female 22.5 to 28 cm. long by 635μ in maximum width. Cuticle transversely striated at 5 to 6μ intervals. Mouth unarmed (Fig. 105), surrounded by 6 small papillae, funnel-shaped, the cuticle thickened at the anterior end. The blunt, conical tail (Fig. 106) curves in a weak spiral. Anus 155 to 250μ from the tip of the tail. Vulva 1.1 to 1.16 mm. from the head end. Vagina 5 mm. long. Two uteri and ovaries form numerous loops, almost filling the body cavity. Ovoviviparous. Eggs (Fig. 107) 46μ long by 39μ wide, bluntly ellipsoidal. These hatch in the uterus, the vagina and distal part of uteri containing embryos.

Embryo (Fig. 108) 290μ long by 11μ wide, with blunt head and a pointed tail bent in a hook. Intestine clearly defined.

Life history.—Unknown; presumably there are stages in an intermediate host which may be looked for among the biting arthropods.

Distribution.—Indo-China (Hué) and Dutch East Indies.

Pathology.—This worm gives rise to thrombi in the pulmonary blood vessels, the thrombus surrounding portions of the lumen and causing the vessels to be sometimes markedly contorted. The vessel walls are inflamed and surrounded by cellular infiltration. There are local pneumonic areas, edema and hemorrhagic infiltration. No clinical symptoms have been noted.

Treatment.—Unknown.

Prophylaxis.—Unknown; measures to guard against attacks of biting arthropods are indicated.

Although it would appear probable that the microfilaria of Bauche and Bernard (page 129), found in the blood of swine in Annam, was

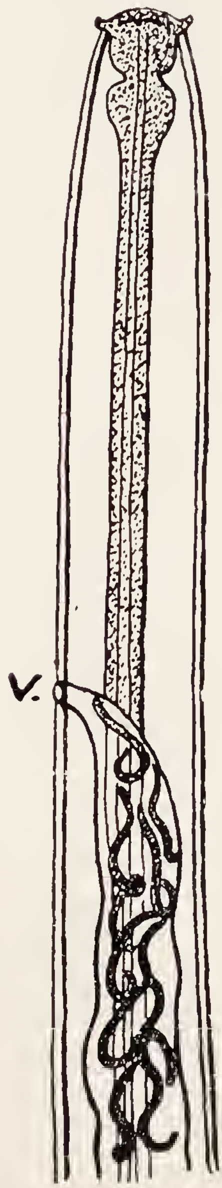


Fig. 105. *Filaria bauchei* (*F. hellemansi*). Anterior end of female. Enlarged. From Smit, 1920. *v*, vulva.

Fig. 106. *Filaria bauchei* (*F. hellemansi*). Female tail. Lateral view. From Smit, 1920.

the larva of *F. bauchei*, it would be unsafe at this time to make any assertion in view of the marked difference in their size and the size of the embryos of *F. bauchei*.

Fig. 107. *Filaria bauchei* (*F. hellemansi*). Eggs. Enlarged. From Smit, 1920.

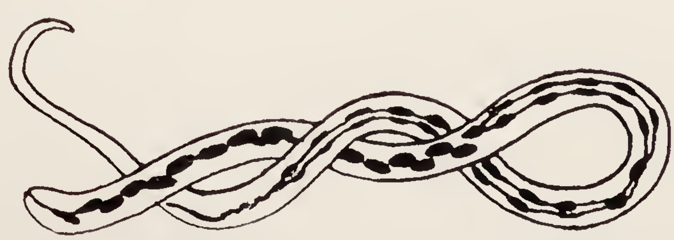


Fig. 108. *Filaria bauchei* (*F. hellemansi*). Embryo. Enlarged. From Smit, 1920.

MICROFILARIA SP. of Bauche and Bernard, 1912

Microfilarid of swine

Synonym.—See discussion of *Filaria bauchei*, page 128.

Hosts.—Primary: Swine; secondary: Unknown, probably biting arthropods, especially Diptera (flies).

Location.—In circulating blood as embryos.

Morphology.—*Microfilaria*: First stage larvae without sheath, 90 to 110μ long. Head abruptly rounded. Body tapers slightly through the posterior fifth to the tip of the tail, which is slightly rounded. Stained specimens 54 to 96μ long by 4μ wide. Interior of body granular except at 3 places, (1) a cephalic area, V-shaped, with the point directed posteriorly, (2) a ring-shaped area almost perpendicular to the body axis about 26/100 of distance from anterior end, and (3) an oval area 37/100 of distance from anterior end.

Life history.—Unknown; the adults presumably live in swine outside of the digestive tract and circulating larvae in blood are probably taken up by biting arthropods in which they undergo some development and are then transferred to swine by biting them.

Distribution.—Central Annam.

Pathology.—Unknown.

Treatment.—Unknown.

Prophylaxis.—Unknown; prevention of attacks by biting arthropods is indicated.

McNaughton has reported a microfilaria from the blood of swine in the Ellice Islands, but has given no description.

SETARIA BERNARDI Railliet and Henry, 1911

The setaria of swine

Synonyms.—*Setaria sp.* Gedoelst, 1911; *Filaria papillosa* var. *suis* Smit, 1918; *Filaria labiato-papillosa* var. *suis* Smit, 1923.

Hosts.—Primary: Swine; secondary: Unknown, probably some biting arthropod.

Location.—Peritoneal cavity and encysted on the surface of liver.

Morphology—*Setaria*:

Male 7.5 to 11 cm. long by 650μ in maximum width. Tail twisted in a loose spiral, with a pointed end, and with 8 pairs of papillae, of which 4 are preanal and 4 postanal. Smit's figure (Fig. 109) shows 3

postanal and 5 preanal, but interpretations in this connection may vary with different observers. Spicules unequal; the longer 215μ long by 25μ wide, with a membranous extension 70μ long; the shorter 140μ long by 52μ wide.

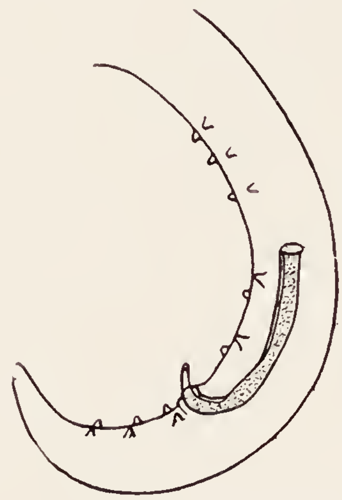


Fig. 109. *Setaria bernardi.* Male tail. Enlarged. From Smit, 1920.

Female 20 to 21 cm. long. Anus 300μ from the tip of the tail. Vulva 600μ from the head end. Eggs ovoid, 45μ long by 26μ wide. Ovoviviparous.

Life history.—Unknown; probably involves intermediate stages in some biting arthropod.

Distribution.—Indo-China (Hué) and Batavia.

Pathology.—Unknown.

Treatment.—Unknown; probably no treatment possible, or, as a rule, necessary.

Prophylaxis.—Unknown; as a tentative measure, prevention of attacks by biting insects is indicated.

DIOCTOPHYME RENALE (Goeze, 1782) Stiles, 1901

The giant kidney worm

Synonyms.—See Parasites of Dogs.

Hosts.—Primary: Swine, rarely, dogs and wild carnivores, usually (See Parasites of Dogs); secondary: Fish (*Idus idus*), according to Ciurea.

Location.—Kidney of swine, and kidney, abdominal cavity and rarely elsewhere in other primary hosts; in tissues of secondary hosts.

Morphology.—*Dioctophyme*: Very large blood-red worms; the male 14 to 40 cm. long by 4 to 6 mm. wide and the female 20 to 102 cm. long by 5 to 12 mm. wide.

Life history.—The eggs from female worms in the kidneys pass in the urine and apparently infect fish in some manner when the eggs get in water in which there are suitable fish hosts. Presumably the larvae developing in these fish are capable of infecting suitable primary hosts when infested fish are eaten by these hosts. At present the life history outlined here is based on the finding by Ciurea of a larva which he regards as that of this worm occurring in a fish and the finding of a worm in a dog fed on this fish.

Distribution.—United States, South America, Europe, Asia and Africa.

Pathology.—The worms in the kidneys destroy the kidney tissue. Little is known of this condition in swine as the parasite is rare in this host. The condition is best known for carnivores (See Parasites of Dogs).

Treatment.—None known.

Prophylaxis.—Prevent swine from eating raw or uncooked fish.

TRICHURIS SUIS (Schrank, 1788) A. J. Smith, 1908

The whipworm of swine

Synonyms.—*Trichocephalus suis* Schrank, 1788; *Trichocephalus apri* Gmelin, 1790; *Trichocephalus crenatus* Rudolphi, 1809.

Hosts.—Swine, wild boar and wild pig (*Sus bengalensis*).

Location.—Cecum and colon, the anterior portion embedded in the mucosa.

Morphology.—*Trichuris*: The anterior portion of the body long and slender, the posterior portion short and thick. Esophagus a chitinous tube embedded in a single layer of cells.

Male 3.3 to 4 cm. long, the anterior portion of body constituting 5/8 of the entire length. The single spicule (Fig. 110) is 3 mm. long, rounded at the end; its sheath (Fig. 111) is bell-shaped and bears numerous blunt spines. In this worm and in related worms the ejaculatory duct opens by a latero-dorsal aperture into the cloaca (Fig. 112), whereas in most nematodes, according to Rauther, this aperture is ventral.

Female 3.4 to 5 cm. long, the anterior portion of the body twice as long as the posterior portion. There is a single ovary and uterus (Fig. 113). Eggs lemon-shaped, 52 to 56μ long, and brown.

Life history.—Apparently simple. The eggs pass in the manure of the host and embryos develop in them. When swallowed by swine,

these young worms develop to adults, probably directly and without migration. The development within the body of the host has not been investigated.

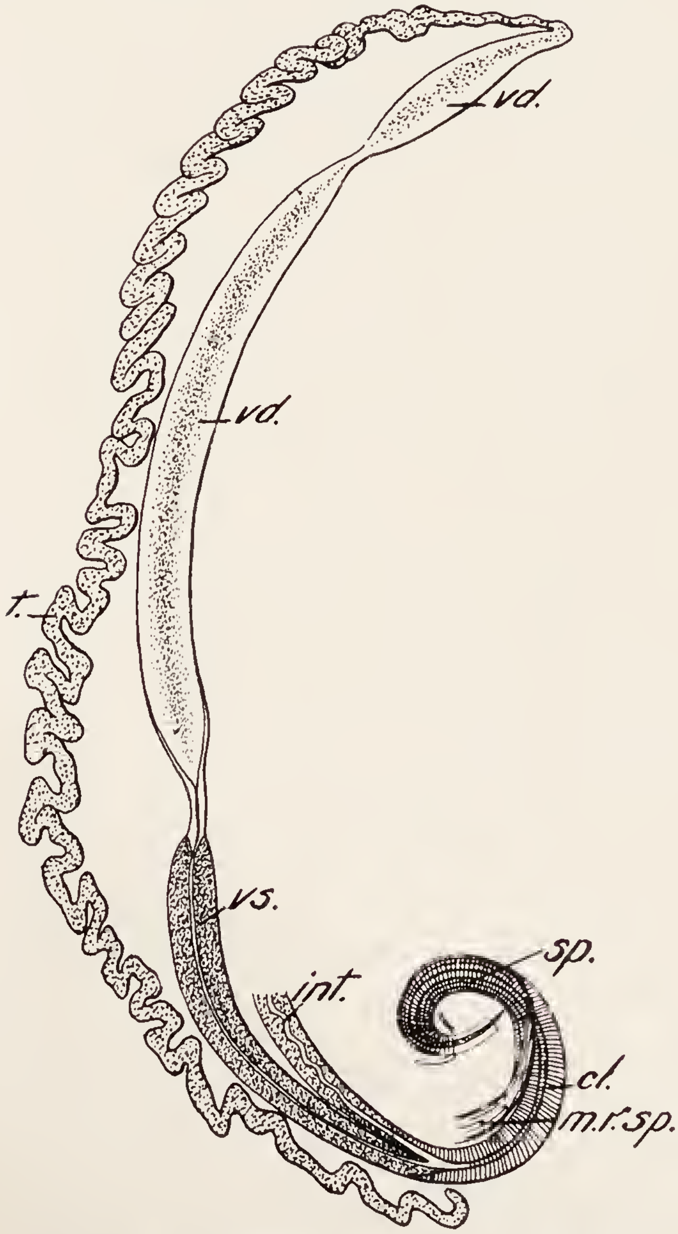


Fig. 110. *Trichuris suis.* Male genitalia. x 11. From Rauther, 1918. *t,* testis; *vd,* vas deferens; *vs,* vesicula seminalis; *int,* intestine; *sp,* spicule; *cl,* cloaca; *mrsp,* retractor muscles of spicule.

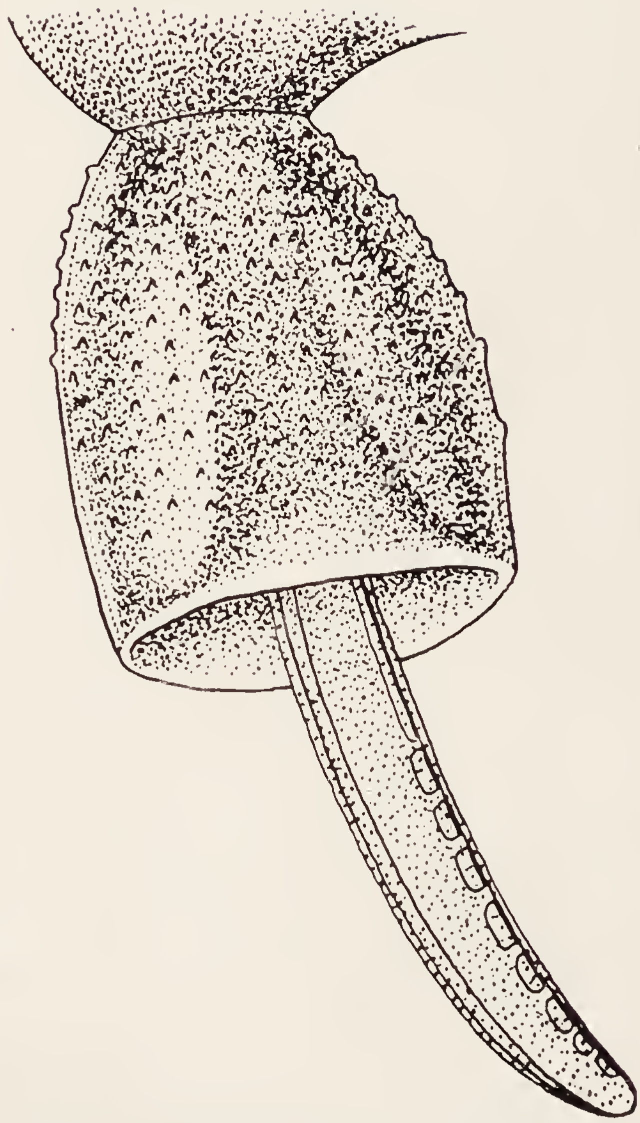


Fig. 111. *Trichuris suis.* Spicule and spicule sheath. x 200. From Schneider, 1866.

Distribution.—Cosmopolitan. Common in the United States.

Pathology.—No symptoms are noted in moderate infestations. Haase has reported cases where young animals have been killed by massive infestations with whipworms. The swine were weak and emaciated. A post mortem showed a marked typhlitis, dry cecal content, and the buccal mucosa and base of the tongue swollen, highly inflamed and covered with a thin fibrinous coating. Bonhill reports that in swine

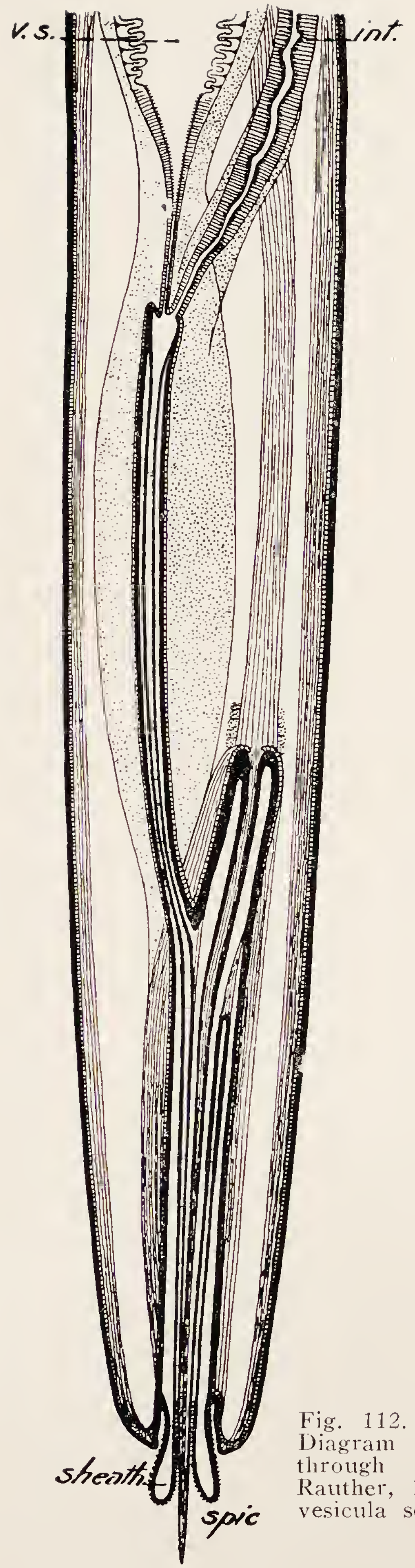


Fig. 112. *Trichuris suis.* Male. Diagram of longitudinal section through posterior end. From Rauther, 1918. *int,* intestine; *vs,* vesicula seminalis; *spic,* spicule.

Fig. 113. *Trichuris suis.* Female genitalia. x 11. From Rauther, 1918. *va.* vagina; *ut,* uterus; *rs,* receptaculum seminis; *ovd,* oviduct; *ov,* ovary.

examined post mortem by him whipworms were present in the young shoats, but not in the older pigs.

Treatment.—Uncertain. It would probably require repeated treatments with some such non-irritant drugs as santonin for a number of consecutive days or a massive dose of some relatively non-toxic drug to remove these worms. Occasional worms may be removed by a single dose of the usual anthelmintics.

Prophylaxis.—Sanitation, such as that recommended for the control of swine ascarids, is indicated as a control measure (See page 60).

TRICHURIS OVIS (Abildgaard, 1795) A. J. Smith, 1908
The whipworm of ruminants

Synonyms.—See Parasites of Sheep.

Hosts.—Swine and, commonly, cattle, sheep, goats, etc. This worm is occasionally reported from swine. It is still questionable as to whether the worms found belong to this species or to *Tr. suis* (Page 131).

Location.—Cecum and colon.

Morphology, life history, etc.—See Parasites of Sheep. This worm resembles *Tr. suis* in a general way. It is not of known importance in connection with swine.

TRICHINELLA SPIRALIS (Owen, 1835) Railliet, 1895
Trichina

Synonym.—*Trichina spiralis* Owen, 1835.

Hosts.—Primary: Swine, rat and man, and, less often, various other animals; secondary: The same, the worm passing its larval stage, as well as its adult stage, in the same host animal.

Location.—In the small intestine as adults; in the voluntary musculature as larvae.

Morphology.—*Trichinella*: Minute worms of nearly uniform diameter throughout, but slightly thicker posteriorly. The round head has an unarmed mouth opening into the tubular portion of the esophagus, which after a short course is imbedded in the single layer of cells present in this and related forms. At the posterior end of the esophagus are 2 appendices. The anus is terminal.

Male 1.4 to 1.5 mm. long by 40μ thick (Fig. 114). The terminal portion of the vesicula seminalis and the cloaca can be extruded in copulation (Fig. 115). Two conical projections on each side of the cloaca, with 4 papillae behind them (Fig. 116).

Female 3 to 4 mm. long by 60μ thick. A single ovary and uterus are present (Fig. 117). The eggs are subspherical and 40 by 30μ in diameter; there is a delicate vitelline membrane, but no true eggshell.

Embryos 100 to 160μ long by 9μ thick, being thicker at the anterior end and decreasing in thickness posteriorly.

Larvae 300μ to 1 mm. long by 30μ thick. Anterior portion thin-

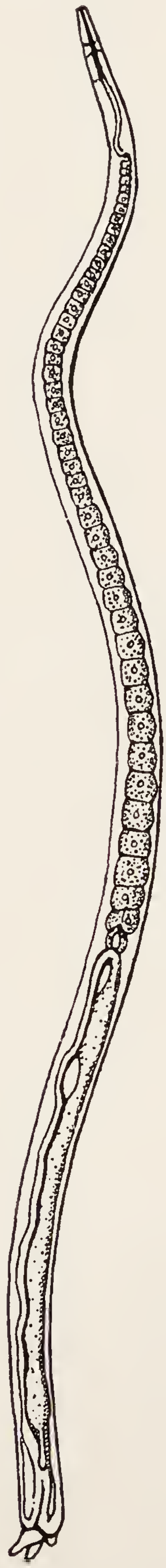


Fig. 114. *Trichinella spiralis.* Male. Enlarged. From Leuckart, 1866.

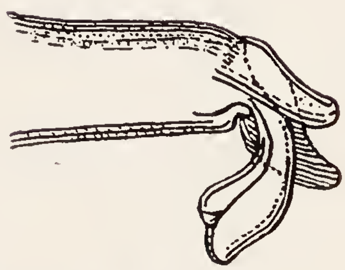


Fig. 115. *Trichinella spiralis.* Posterior end of male, showing extruded cloaca. Enlarged. From Leuckart, 1866.

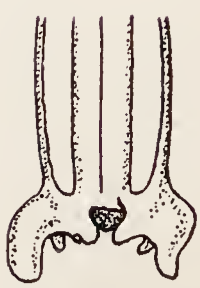


Fig. 116. *Trichinella spiralis.* Posterior end of male, showing clasping projections and papillae. Enlarged. From Leuckart, 1866.

ner and more pointed, the posterior thicker and rounded. The larvae lie in a cyst (Fig. 118) with the ventral surface toward the periphery;

Fig. 117. *Trichinella spiralis.* Female. Enlarged. From Leuckart, 1866.

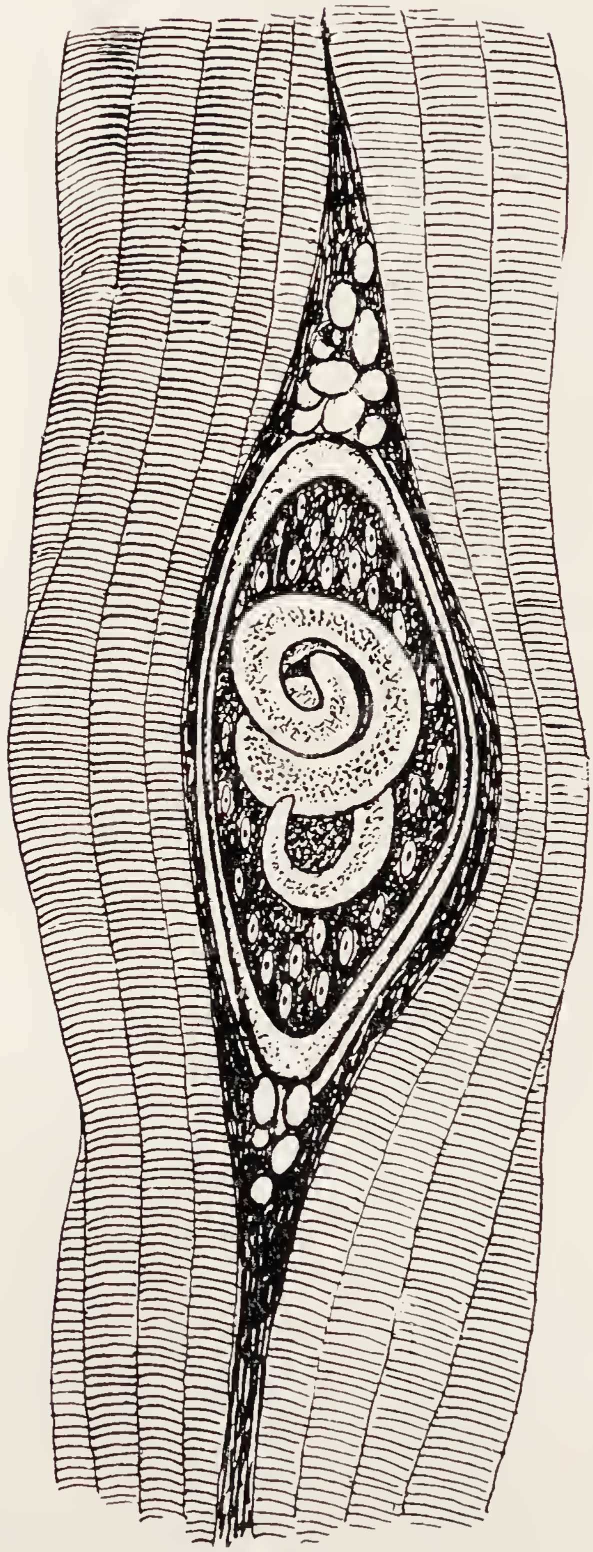


Fig. 118. *Trichinella spiralis.* Larva encysted in a piece of muscle. Enlarged. From Ransom, 1907, after Leuckart.

in the larval female the primordium of the genitalia is on the ventral side of the posterior portion of the esophagus; in the male larva the primordium of the genitalia is on the dorsal surface.

Life history.—The adult male and female worms copulate in the intestine of the host animal. The male then dies, while the female bores into the mucosa, into the glands of Lieberkuehn, and gets into the lymph spaces. Here the embryos are deposited, beginnning about a week after infection, the female living 5 to 6 weeks and bearing 1500 to 1800 embryos (Braun says 10,000). The embryos make their way by the blood and lymph channels throughout the body and come to rest in the voluntary muscles, where they force their way from the intermuscular connective tissue into the sarcolemma. Here they develop to the infective larvae described above. The larva is at first free (Fig. 119), later encapsuled and rolled into spirals, rings or figures-of-eight. When meat containing these larvae is ingested by a suitable host animal, the capsules are digested in the stomach and the worms are set free in the course of 24 hours. The larvae enter the intestine and become sexually mature in 1 to 5 days. There are about 12 females to each male.

Swine become infested by eating carcasses of infested swine or rats, and in turn serve as the source of infestation for man when eaten as under-cooked pork and pork products, such as sausage, Italian hams, lockschinken, and ordinary pork and ham. Trichinae are said to sometimes remain alive in the muscles of swine for as long as 11 years.

Distribution.—Cosmopolitan. Of over 8,000,000 swine inspected microscopically for trichina in the United States during the years 1898-1906, 1.41 per cent contained living trichinae, and an additional 1.16 per cent contained trichina-like bodies or disintegrating trichinae. In other words, 1 out of every 39 swine was infected, and 1 out of every 71 swine was infective for man. Up to 1915, there had been about 1,550 cases of trichinosis in man reported for the United States, according to Ransom, of which about 16 per cent were fatal. Most of the cases are reported from persons of German or Italian nationality, a fact connected with their habit of eating raw or under-cooked pork products in the form of sausage, Italian hams, etc.

Pathology.—In heavily infested experiment animals, the customary clinical picture of trichinosis is seen in swine as in other animals. There is a pyrexia, less marked than in man, occasional edema of the eyelids, and the stiffness and pain on movement, as well as dyspnoea, that accompanies the migration of the embryos through the body. A blood

count shows that there is a marked rise in the eosinophile count. After the migration of the trichinae into the muscles, these larvae may be demonstrated by harpoon puncture of the voluntary muscles and the

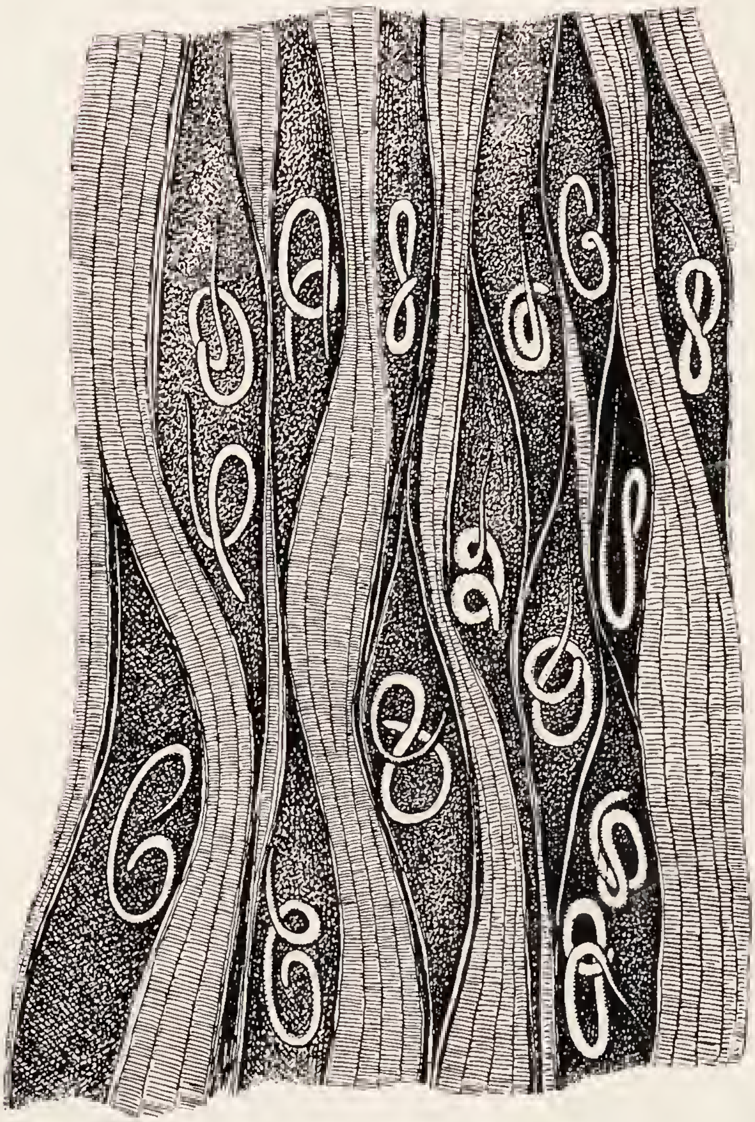


Fig. 119. *Trichinella spiralis.* Larvae in muscles but not yet encysted. Enlarged. From Ransom, 1907, after Leuckart.

examination of the muscle tissue thus removed, either by direct microscopic examination of a press preparation or by digestion of the muscle in artificial gastric juice, centrifuging, and microscopic examination of the sediment for larvae.

In actual practice, trichinosis in swine due to infestation naturally acquired is not diagnosed. Infestation severe enough to cause clinical symptoms marked enough to recognize is rare and swine survive even heavy infestations and show no subsequent bad effects. Eosinophilia is much more apt to be due to the presence of the more common intestinal parasites and so gives little clue to the presence of trichinosis, though very high counts would suggest the existence of trichinosis.

In some countries, as in Germany, trichinosis in swine is of great importance in meat inspection, since in these countries the eating of raw and inadequately cooked pork is a well established national habit. The preferred sites of infestation are the diaphragm, the pillars of the diaphragm, muscles of the larynx, tongue and abdomen, and the intercostals. Usually pieces of the diaphragm are examined in press preparations with a microscope or the trichinascope, the latter instrument being an arrangement whereby the picture of a press preparation is thrown on a screen, giving a considerable enlargement and making the detection of the trichinae somewhat easier and simpler than by means of a microscope.

In the United States, federal inspection of meat for the presence of trichinosis has been abandoned since 1906. Protection against trichinosis in man here has depended on educational campaigns and warnings against the consumption of raw or inadequately cooked pork and pork products and the use of certain measures in federal meat inspection to ensure the safety of pork products that are customarily eaten raw by a suitable treatment of the pork that goes into them. While trichina inspection would eliminate a certain amount of trichinous pork in this country as elsewhere, it would give a false sense of security against trichinosis, as not much over half of the meat production in the United States is under federal supervision, and meats in general would be regarded as safe as regards trichinosis in view of this inspection, a fact that would doubtless contribute to an increase in trichinosis. On the other hand, the advice to cook pork thoroughly is a safeguard against trichinosis from pork of any origin. Furthermore, inspection does not eliminate all trichinous pork from the market and is very expensive. At a cost of 15 cents for this inspection for each carcass, it would add, according to Ransom, $5,000,000 to our 1915 budget expense of over $3,000,000 for meat inspection. Dependence is therefore placed on adequate cooking, refrigeration and processing. Experiments show that larval trichinae die very rapidly when exposed to a temperature of 55 degrees C., but may recover if exposed to a temperature slightly below this for a short time; death or recovery depends on whether the protoplasm has been irreversibly coagulated or not. Prolonged temperatures up to 50 degrees C. kill larvae, apparently as result of exhaustion following excessive activity from heat stimulation. For the purposes of the federal meat inspection, a temperature of 137 degrees F. (58.33 degrees C.) is required as the minimum temperature to which pork and pork products of a sort customarily eaten without cooking must be heated, thus giving a certain

margin of safety above the exact thermal death point. In refrigeration processes, exposure to a temperature of 5 degrees F. (—15 degrees C.) for 20 days ensures the death of the trichinae, apparently as a result of the precipitation of the colloids from the protoplasm and the resultant freezing of certain constituents to the point where they are no longer able to resume their place in the protoplasmic complex. Such frozen trichinae show evidence on microscopic examination of alterations in structure. For temperatures around freezing, trichinae are very resistant. Certain pickling processes, suitably carried out, will also kill trichinae as a result of the action of salt, drying and temperature. Investigations covering meat inspection procedures in relation to trichinosis in the United States have been carried out by Ransom, Schwartz and Raffensperger.

Treatment.—So far as swine are concerned, there is no treatment for trichinae. Whether it is possible to kill the trichinae in the muscles is of little interest, as they are irremovable and act as foreign bodies whether alive or dead, and these worms commonly die and become calcified ultimately. As regards the adult trichinae in the intestine, it is likely that they can be removed without especial difficulty by any one of a number of anthelmintics, such as therapeutic doses of oil of chenopodium (1 dram for a 100-pound animal), turpentine (2 to 4 drams), or santonin (1 to 2 grains, with an equal amount of calomel, daily for a week). However, this removal of the intestinal worms is of value mostly in the early stage of the disease before the females have entered the wall of the intestine to deposit their embryos in the lymphatics, and as this stage is indicated only by digestive disturbances, difficult to distinguish from a number of similar digestive derangements, it is not likely to be diagnosed in time to make such treatment of value.

Prophylaxis.—The prevention of trichinosis in swine is a matter of preventing infection with trichinous meat, as the conveyance of trichinae to swine from feces of rats or swine containing trichinae passing from the intestines is regarded today as of little or no importance. Carcasses of swine dying on farms or elsewhere should be burned or otherwise disposed of so that swine cannot eat them, or thoroughly cooked if they are to be fed to swine. This general rule applies to pork scraps and to pork in garbage or in slaughterhouse waste. Rat control is also of value, and measures against rats on farms are indicated to prevent swine catching rats or eating rats that have died from various causes, as well as to prevent the enormous losses due to food consumed and damage done by these rodents.

CHAPTER IV

Acanthocephalids Infesting Swine

MACRACANTHORHYNCHUS HIRUDINACEUS

(Pallas, 1781) Travassos, 1917

The thorny-headed worm of swine

Synonyms.—*Taenia hirudinacea* Pallas, 1781; *Echinorhynchus gigas* Bloch, 1782; *Gigantorhynchus gigas* (Bloch, 1782) Hamann, 1892; *Gigantorhynchus hirudinaceus* (Pallas, 1781) Railliet, 1893.

Hosts.—Primary: Swine and also man, wild boar, hyena, and various Canidae, Felidae, primates, etc.; secondary: Beetles (*Cetonia aurata*, *Lachnosterna arcuata* (*Lachnosterna*=*Phyllophaga*), *Melolontha melolontha* and *Diloboderus abderus*, the May beetles or June bugs being the hosts thus far reported for the United States. Stiles has suggested that *Lachnosterna dubia* and *L. hirticula* may also serve as hosts in the United States.

Location.—The small intestine, especially the duodenum, and, rarely, the large intestine, the head attached to and imbedded in the intestinal wall (reported once from the gall bladder), and, according to Kovarzik, the stomach of primary host; body cavity of secondary host.

Morphology.—*Macracanthorhynchus*: Large worms (Fig. 120). The acanthocephalids are not nematodes but the group is commonly attached to the nematodes. No digestive tract is present in the acanthocephalids. In this species the body is milk-white to bluish, elongate cylindrical, gradually tapering toward the posterior extremity. The cuticle is transversely annulated and often shows moderate swellings. The head has a cylindrical proboscis which can be invaginated or exserted, somewhat swollen at the end, and bearing 5 rows of strong hooks projecting backward (Fig. 121); these hooks are completely covered with a chitinous investment and have 2 projecting roots each. The proboscis withdraws into a sheath from the base of which 2 sacs, the lemnisci, extend into the body cavity. From the sheath there also extends a suspensory ligament which bears the sexual organs.

Male 6 to 10 cm. long by 3 to 5 mm. wide. The posterior end is straight and blunt. There are 2 testes with their vasa deferentia, a

common ejaculatory duct, and a finger-shaped cirrus, 9 mm. long, which may be protruded from the posterior end or retracted into a pouch, commonly termed a bursa (Fig. 122).

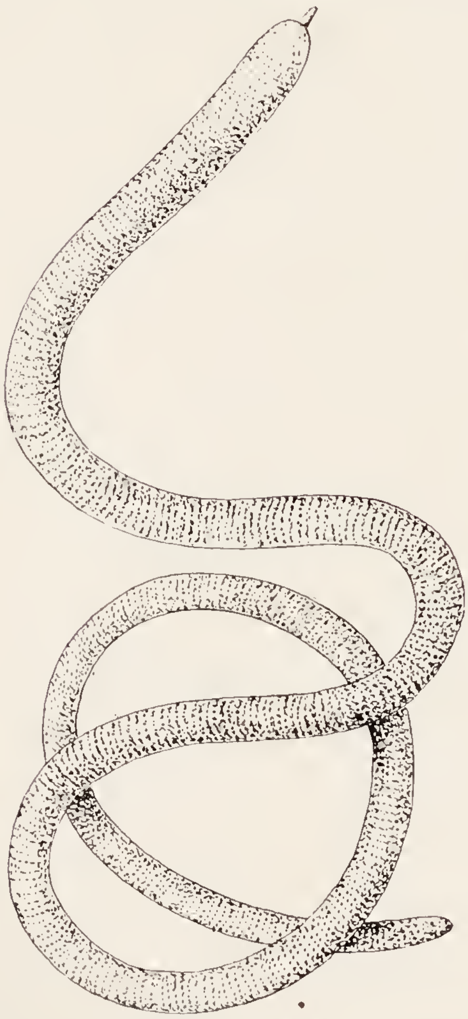


Fig. 120. *Macracanthorhynchus hirudinaceus.* Female. Entire worm. Two-thirds natural size. From Neveu-Lemaire, 1912.

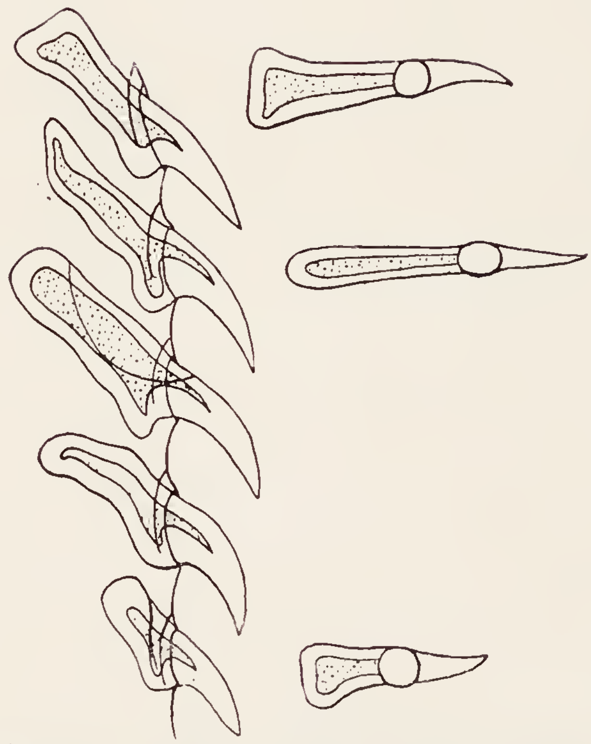


Fig. 121. *Macracanthorhynchus hirudinaceus.* Hooks. Enlarged. From Travassos, 1917.

Female 20 to 35 cm., rarely to 47.5 cm., long by 4 to 9 mm. wide. Two ovaries present, which first lie in the suspensory ligament. The eggs are released to the body cavity and are then taken up by the uterus, after fertilization in the body cavity, passing in through the uterine bell. They pass to the exterior through a short vagina (Fig. 123). The eggs are 90 to 110μ long by 51 to 56μ wide, and oval (Fig. 124); the embryo is surrounded by 3 shells of which the middle is the thickest. The embryo is coiled up and has at the anterior end 4 large hooks and a number of smaller ones, the entire body being covered with small spines.

Life history.—Schneider in 1868 first found an intermediate host of this worm in *Melolontha vulgaris.* In 1887 Kaiser incriminated *Cetonia aurata,* and in 1891 Stiles showed that *Lachnosterna arcuata* and possibly

L. dubia and *L. hirticula* would act as hosts. The eggs pass out in the feces of the host and may be swallowed by the "white grubs" or May beetle larvae, or other beetle larvae, as they feed in manure, or in soil mixed with manure, from infested swine. In the beetles the young worm escapes from its shell and bores through the intestinal wall to

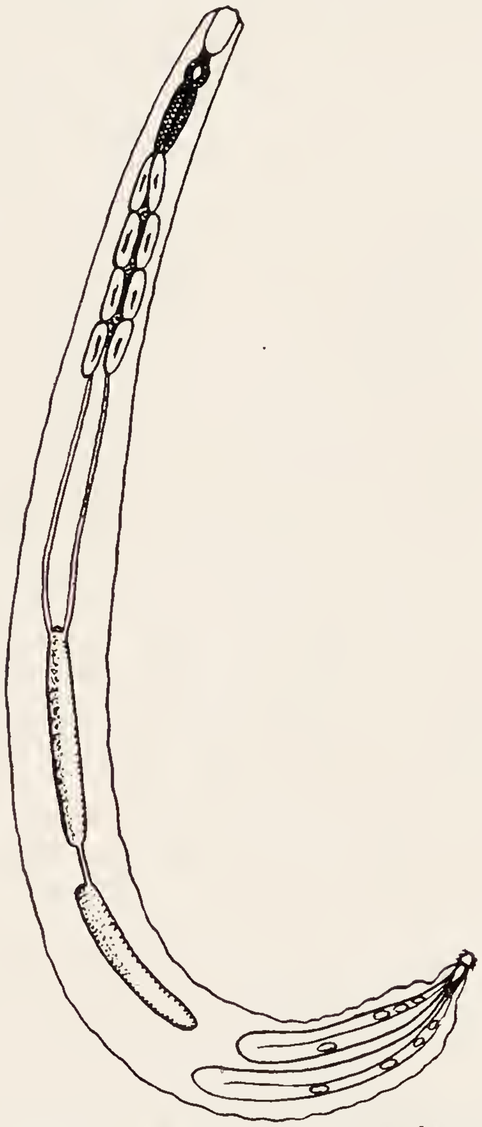


Fig. 122. *Macracanthorhynchus hirudinaceus.* Adult male. Enlarged. From Travassos, 1917.

Fig. 123. *Macracanthorhynchus hirudinaceus.* Female ovejector. Enlarged. From Travassos, 1917.

the body cavity of the beetle, where it encysts. In the metamorphosis of the beetles to adults, the encysted worms remain in the body cavity. When swine eat the larvae, as they are prone to do in rooting through areas where the beetle larvae occur, or when they eat the adult, as may readily happen when the May beetles alight where swine are feeding, the insects digest and release the worms, which develop to maturity in the intestine.

Distribution.—Cosmopolitan (North and South America, Europe, Africa, Asia, Australia, and Madagascar); common in the United States.

Pathology.—At the site of attachment in the intestine, these worms set up an inflammatory reaction with some necrosis, the site showing from the exterior as a swelling or nodule. Occasionally perforation of the intestine occurs, with a resultant peritonitis. As a rule these worms are not ranked as of major importance, but they are regarded as injurious beyond question. Steffani reports finding this worm in the gall bladder, and Kovarzik reports it from the stomach in 2 pigs dying

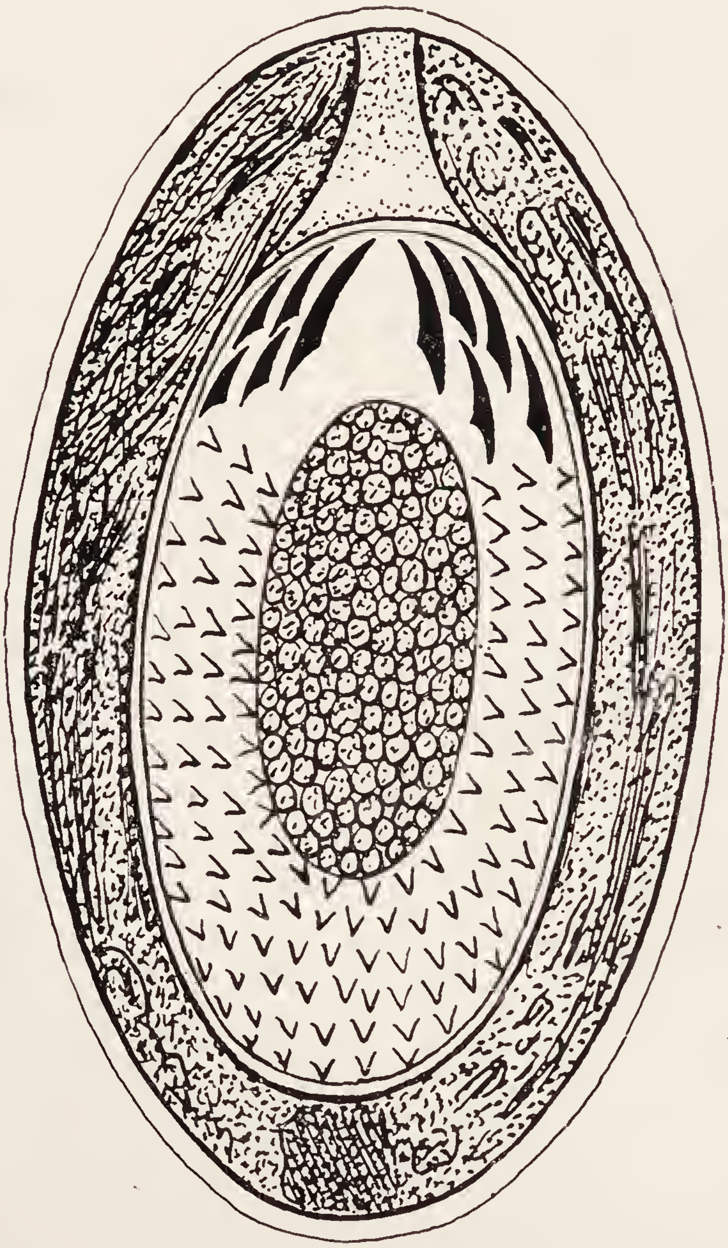


Fig. 124. *Macracanthorhynchus hirudinaceus.* Egg. Enlarged. From Ward, 1907, after Braun.

in a herd of 50 infested and showing clinical effects. No special symptoms have been definitely associated with these worms, the effects in the production of unthrifty animals with digestive disturbances and without fever being the customary effects of intestinal parasites in gen-

eral. Alessandrini reports that this worm contains a hemolysin of a colloidal nature, water soluble, but not alcohol-soluble, the action of which diminishes at 40 degrees C. and is lost at 55 degrees C.

Treatment.—Little in the way of critical experiment has been done on anthelmintics for removing this worm, though a number of recommendations are made in the literature. Turpentine in doses of 1 tablespoonful (4 drams) in a light feed or in skim milk for 3 successive feeds has been recommended for a 100-pound animal, and powdered copper sulphate in doses of 1 teaspoonful (1 dram) in a thick mash morning and evening has been recommended, either treatment to be preceded by fasting and followed by 1 ounce of Epsom salts for animals of the size mentioned. In the absence of critical tests, oil of chenopodium, in doses of 1 dram for animals weighing 100 pounds, given in 2 ounces of castor oil, would be worth trying. Calandruccio reported the removal of 53 echinorhynchs, *Moniliformis moniliformis*, from himself with no evidence of worms present subsequently by means of 8 grams of ethereal extract of male fern. This drug might prove of value in removing the thorny-headed worms of swine. It has been reported that carbon tetrachlorid will remove these worms, but we lack definite information as to the efficacy of this drug in this connection. Experiments carried out by the United States Bureau of Animal Industry have indicated that carbon tetrachlorid would remove a few thorny-headed worms and leave a larger number. However, Kovarzik reports the successful treatment of an infested and diseased herd by means of a Hungarian proprietary remedy, Vermithan, containing carbon tetrachlorid and isobornylacetate, the animals recovering and taking on weight. One pig died 2 days after treatment and the thorny-headed worms in it were found dead and distorted.

Prophylaxis.—Keeping swine from rooting over areas on which or near which swine have been pastured is a preventative measure, though not a complete safeguard as beetles, which, however, are much less likely to be eaten than are the grubs, may fly considerable distances, carrying the infective larvae in their bodies. Forbes reports that 108 pigs ate over 99 per cent of the grubs in a badly infested 10-acre corn field in 27 days, a number calculated at 1,217,083 grubs, or 11,278 grubs per pig. Had these grubs been generally infested with the larvae of thorny-headed worms serious injury would probably have followed. Measures for the destruction of white grubs are also valuable and warranted by the damage which these grubs do to plants. Some of these measures are given by Davis as follows: Allow poultry to follow the plough, harrow and cultivator and destroy the grubs turned up. Use

portable poultry houses in the fields. If hogs are used to destroy grubs use them on fields not previously pastured by hogs within 3 years. Use a selected crop rotation, such as oats or barley, followed in turn by clover and then corn. Fall plowing before the grubs go deep into the ground to pass the winter aids in destroying grubs, as do summer and fall plowing when the grubs are changing to beetles. This should be followed by deep disking to break the soil. Adult beetles may be caught by jarring them onto cloths from the plants on which they feed at night or may be trapped at lights. An important brood of these beetles matures in the United States every 3 years, in 1914, 1917, 1920, 1923, etc. Spraying the food trees with arsenicals is effective, but rarely practical.

CHAPTER V

Linguatulids Infesting Swine

LINGUATULA SERRATA Froelich, 1789

The tongueworm

Synonyms.—*Taenia rhinaria* Pilger, 1802; *Linguatula rhinaria* (Pilger, 1802) Railliet, 1885. For additional synonyms see Parasites of Dogs.

Hosts.—Primary: Dog, wolf, fox, horse, mule, sheep, goat and man; secondary: Swine, cattle, sheep, goat, deer, antilope, dromedary, horse, peccary, rabbit, hare, guinea pig, cat and man.

Location.—Nasal fossae or, rarely, frontal sinus as adults; viscera, especially the liver, lungs and lymphatic glands, as larvae.

Morphology.—*Linguatula*: These are not true worms, but are regarded as degenerate arachnids, relatives of the spiders, ticks, etc. They are included here because they are often regarded as worms, commonly studied by helminthologists, and given little or no consideration in most books on veterinary entomology.

For morphology of the adult parasite, see Parasites of Dogs.

Larva. The fully developed infective larva (Fig. 125) is 4 to 6 mm. long by 1.2 to 1.5 mm. wide, lanceolate in outline and whitish in color. It has 80 to 90 annulations, each annulation having a median series of stigmata and bearing on its posterior margin a series of spines directed posteriorly. The mouth is elliptical and surrounded by the 4 characteristic teeth and the accessory hooks. The digestive tube is large. The genitalia are rudimentary, but the sex may be determined by the position of the genital orifices.

Life history.—The eggs are expelled from the nostrils of the host in sneezing and probably escape to some extent in the feces, though this would appear to involve the likelihood of infesting the primary host with the larval stages, a thing which occurs at times. It is possible that the carnivore hosts of the adult tongueworm have some degree of immunity to infestation with the larva, as the escape of the eggs from the nostrils must subject these hosts to infestation with the larval worms to a large extent as a result of contamination of food and water.

When these eggs are swallowed by a suitable host, the eggs hatch and the young larvae traverse the wall of the digestive tract and encyst in the mesenteric lymph glands, liver, lungs, etc. They lose their perforating apparatus and legs and molt twice in the course of 8 weeks, the larva at the eighth week lying in a cyst with the 2 molted cuticles. This larva is 250 to 500μ long by 180μ wide and lies curled up in the

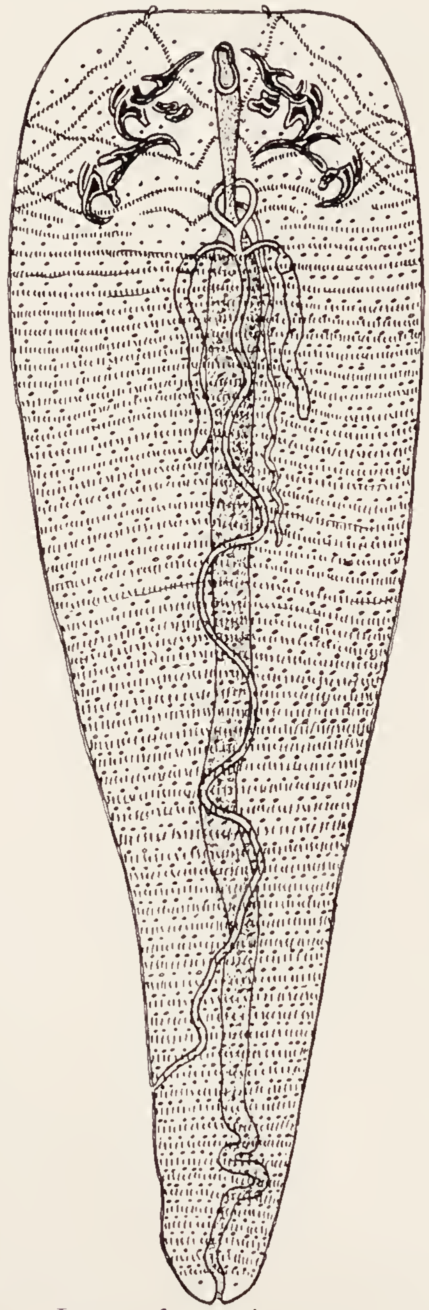


Fig. 125. *Linguatula serrata.* Larva from liver of horse. Enlarged. From Fiebiger, 1923, after Csokor.

cyst; it is without segments, hooks or spines, but has on its dorsal surface 3 rows of apertures, each connecting with a uni-cellular gland, and has a complete digestive canal. About the ninth week the larva molts a third time, and then has 10 to 12 rows of glandular apertures and the primordia of the nervous and genital systems. There are 3 other molts

in the next few weeks, and in about 6 weeks the larva is 1.2 mm. long and signs of segmentation, corresponding to the rows of glandular apertures, appear, together with the peribuccal hooks. In 5 to 6 months, and after 3 more molts, the larval development to the infective stage is complete.

According to Koch, larvae may travel by the lymphatics to the mesenteric lymph glands or via the thoracic duct to the lungs, or by the blood stream to the liver or may lodge under the intestinal serosa or in the spleen or kidneys.

These larvae may not remain encysted at the original place of encystment. After a time they may rupture their cyst and fall into the pleural or peritoneal cavity. According to Railliet, Babes has ascertained that larvae encysted in the mesenteric lymph glands or the wall of the intestine may enter the lumen of the intestine and pass out in the feces. Larvae in the lungs may enter the bronchi and be found free there, and Railliet notes that in this manner they might reach the nasal cavity of the host and develop there to maturity, thus explaining the occurrence of these tongueworms as adults in the nostrils of herbivores.

When the infective larvae are swallowed by a dog or other carnivore host of the adult tongueworm, the larvae make their way to the nostrils and become mature adults. The exact method by which they reach the nostrils is not yet definitely known. It has been surmised that the larvae might reach the nasal passages through the guttural orifices or even by the nares, but apparently the larvae usually reach the stomach in cysts and are there liberated by the digestion of the cyst. Gerlach thinks that the larvae traverse the wall of the intestine and the diaphragm, enter the lung tissue and then the air passages of the lungs, ascending these latter to the nasal cavities. There is also the possibility that larvae might return to the pharynx by way of the esophagus and so reach the nostrils. After reaching the nostrils, the larvae molt once more in the course of 3 weeks, rapidly growing in size and mating in 6 to 7 weeks. The male reaches its full size in the fourth month and the female in the sixth. They may live in the nostrils for 15 months or for several years. Schornagel found them least common in dogs from October through January.

Distribution.—North America (United States; adult reported from dog at Atlanta, Georgia; larvae common in livers of Southern cattle), Central America (Nicaragua), South America (Brazil), India, Dutch Indies, Australia and Europe.

Pathology.—The young larvae in the liver may be detected as whitish spots just under the capsule. In the lymph glands they form cav-

ities full of brownish material in some cases and in others the glands may be indurated or calcareous. According to Railliet the larvae have little effect on the health of the host. In a case reported by Landon, a man with an infestation with the adult tongueworm in the nostrils had previously suffered from pains in the liver, with icterus and intestinal disorders, and these have been regarded as due to the presence of larval worms in the liver. According to Koch, massive infestations may prove fatal, causing hemorrhages, peritonitis and pleurisy.

Treatment.—There is no treatment of a practical sort for infestation with the larval tongueworms and they are practically always found only postmortem and not antemortem. For the adult tongueworms Railliet states that the treatment should consist in the injection of a parasiticidal substance into the nostrils. He does not suggest such a substance and unless it were very certain in its action it would appear that drug treatment was not indicated and that the animal should be killed as a carrier of a parasite capable of transmission to man, therefore too dangerous to keep as long as there might be any question as to the existence of an infestation. Pillers states: "Antiseptic and irritant solutions are sometimes injected with a view to dislodging the parasites. The results are, however, far from satisfactory. Benzine and weak solutions of ammonia are most often used."

Prophylaxis.—Viscera of meat animals infested with larval tongueworms should be tanked in slaughter-house inspection and effectively destroyed where animals are slaughtered outside of an adequate meat inspection service. Dogs infested with the adult tongueworms, as noted above, should be killed unless the removal of the tongueworms can be ensured.

ARMILLIFER ARMILLATUS (Wyman, 1848) Sambon, 1922

The braceleted porocephalid

Synonyms.—*Linguatula armillata* Wyman, 1848; *Pentastomum moniliformis* Diesing, 1835; *Pentastomum constrictum* von Siebold, 1852; *Porocephalus armillatus* (Wyman, 1848) Stiles, 1893.

Hosts.—Primary: Snakes (pythons and puff adders); secondary: Swine, dog, and, rarely, man, and also monkeys, lion, leopard, banded ichneumon, Aard wolf, black rat, South African reed buck, giraffe, etc.

Location.—In lungs and trachea of primary host; in liver, peritoneum, lymph glands, mesenteries, walls of digestive tract, lungs, and under capsule of spleen and kidneys of secondary host.

Morphology.—*Armillifer*: Adult parasites (Fig. 126) with elongate body, gradually tapering posteriorly and with a series of annulations. Near the anterior end is a circular mouth in the middle of a series of 4 hooks disposed in an arc.

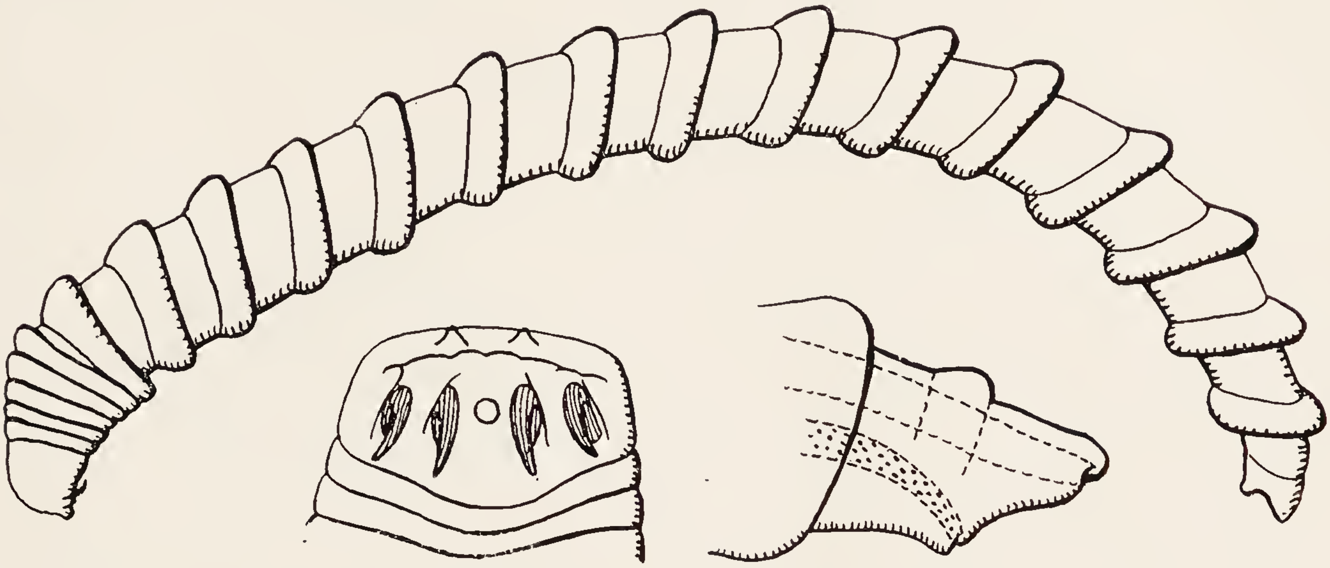


Fig. 126. *Armillifer armillatus.* Above, entire worm, lateral view; lower left, head, ventral view; lower right, female tail showing intestine dorsal to utero-vagina. Enlarged. From Sambon, 1922.

Male 30 to 45 mm. long by 3 to 4 mm. wide, with 16 to 26 annulations. Genitalia consist of a single elongate testis arising near the posterior end of the body and extending dorsad of the intestine to the union of the anterior and middle thirds of the body length; here it divides to form 2 short tubes which unite to form the vesicula seminalis. This divides to form 2 tubes which circle the intestine and continue as the vasa deferentia, which in turn connect with the cirrus pouch. The latter contains a chitinous spicule.

Female 90 to 130 mm. long by 5 to 9 mm. wide, with 18 to 23 annulations. A single ovary occupies a position corresponding to that of the male testis and divides anteriorly to form 2 oviducts which encircle the intestine and open into the seminal receptacles; these open into the vagina by a thick papilla. The vagina is very long. Eggs 108μ long by 80μ wide, contained in a transparent bladder or shell 144μ in diameter.

Embryo (Fig. 127) 92μ long, with 4 ambulatory appendages, each terminating in a pair of claws, and with a perforating apparatus anteriorly and a claw-like tail process posteriorly.

Larva (Fig. 128) similar to adult, but varying in size according to state of development and always found closely coiled in a more or less complete circle with the ventral surface usually forming the convexity of the curve.

Life history.—Similar in general to that of *Linguatula serrata* (See page 148). The young larvae apparently travel in the secondard host to the site of infestation by way of the blood or lymphatic vessels. The

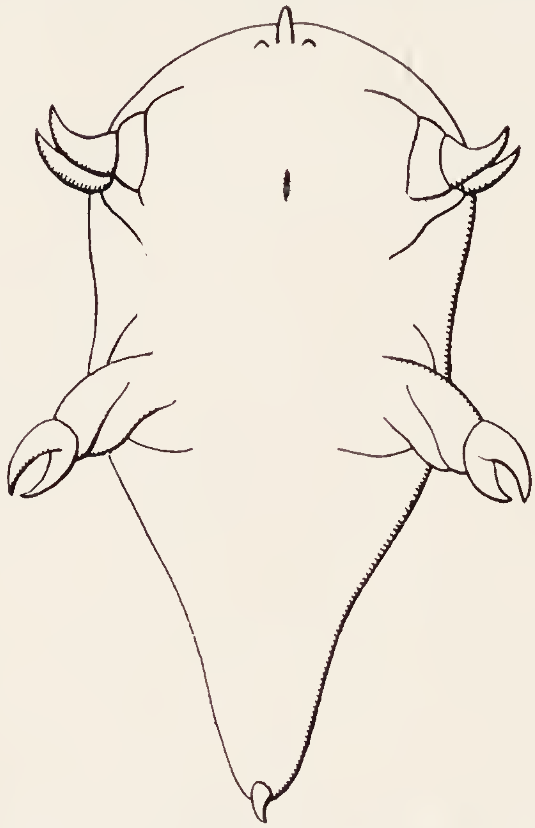


Fig. 127. *Armillifer armillatus.* Embryo. Enlarged. From Sambon, 1922.

Fig. 128. *Armillifer armillatus.* Nymph encysted in host tissue. Natural size. From Sambon, 1922.

eggs of the adult porocephalid may be swallowed in contaminated food or water or ingested in eating a host infested with the adult parasite. According to Noc the egg may hatch in the primary host and the larvae develop in this host.

Distribution.—Africa.

Pathology.—Little seems to have been reported in connection with the pathological aspects of infestation with this porocephalid. Presumably it causes much the same damage as does the larval tongueworm in the same organs and tissues. Noc reports a hypertrophy of the liver in connection with a heavy infestation. The larvae cause a local connective tissue proliferation with a resultant capsule formation and sometimes cause a leucocytic infiltration.

Treatment.—No practical treatment is known for infestations with these larvae.

Prophylaxis.—The destruction of snakes is indicated as a control measure for this parasite, the destruction of these hosts in the case of the pythons and puff adders being desirable on more obvious and weighty grounds than their importance as carriers of parasites.

ADDENDUM

CLADORCHIS GIGANTEUS (Diesing, 1836) Fischoeder, 1901

The large amphistome of swine

Synonyms.—*Amphistoma giganteum* Diesing, 1836; *Stichorchis giganteus* (Diesing, 1836) Fischoeder, 1901.

Hosts.—Primary: Swine, probably accidentally, and peccaries (*Dicotyles albirostris* and *D. torquatus*), usually; secondary: Unknown, but presumably snails. Recently reported from swine by Travassos.

Location.—Large intestine and cecum.

Morphology.—*Cladorchis*: This fluke is 1 to 2 cm. long, elongate oval (Fig. 129), the anterior end narrowed and the posterior portion of body widened and rounded. The cuticula is thick, 28 to 32μ, and shows transverse folds. Oral sucker circular, provided with a sphincter and with paired evaginations or pharyngeal pouches, half as long as the pharynx which projects from the sucker. Pharynx almost spherical, with a sphincter composed of 2 consecutive, concentric rings. Ceca long and slightly wavy, ending postequatorial and posttesticular in acetabular zone. Acetabulum 3.4 to 4 mm. in diameter, oval, terminal

Fig. 129. *Cladorchis giganteus.* Ventral view. x 3. From Fischoeder (1903).

and ventral, slightly sunken, and with large circular aperture. Genital pore 6 to 7 mm. from the anterior end of body, and provided with a sucker 1 to 1.5 mm. in diameter; sphincter present in sucker. No ven-

tral pouch present. Excretory pore apparently postvesicular, in acetabular zone caudad of opening of Laurer's canal. The 2 testes are much branched, about as large as the acetabulum, and are preovarial, near the venter, in the equatorial third of the body; they are arranged tandem and overlap somewhat. Cirrus pouch small, 1 to 1.5 mm. long. Ovary and shell gland almost entirely posttesticular. Vitellaria extend from the zone of cecal bifurcation to the postcecal zone. Uterus intercecal, mostly in testicular zone, with the terminal portion anterior to the testes. Laurer's canal entirely prevesicular, opening at the level of the ovary. Eggs 145 to 156μ long by 72 to 80μ wide.

Life history.—Unknown; perhaps somewhat similar to that of *Paramphistomum cervi* (See Parasites of Cattle).

Distribution.—Brazil.

Pathology, treatment, etc.—Unknown.

INDEX

The object of this index is to enable the reader to find parasites under various names, including the commoner synonyms, to cover intermediate and primary hosts other than swine, to give records of distribution, to furnish a guide to clinical symptoms and pathological conditions, and to list drugs used in treatment.